CAUSES AND PREDICTION OF READMISSION OF HEART FAILURE PATIENTS

Dissertation submitted to

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CHENNAI

APRIL 2015

CERTIFICATE

This is to certify that the dissertation titled **"CAUSES AND PREDICTION OF READMISSION OF HEART FAILURE PATIENTS"** is the bonafide work of **Dr .J SUDHA MALLIKA** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in APRIL 2015. The Period of study was from April 2014 to September 2014.

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DECLARATION

I, **Dr. J SUDHA MALLIKA** solemnly declare that dissertation titled **"CAUSES AND PREDICTION OF READMISSION OF HEART FAILURE PATIENTS"** is a bonafide work done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 during April 2014 to September 2014 under the guidance and supervision of my unit chief **PROF.S.RAJASEKARAN, M.D.,** Professor of Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

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CONTENTS

CAUSES AND PREDICTION OF RE-ADMISSIONS OF HEART FAILURE PATIETS

Sudha mallika J[1], Rajasekaran S[2]

OBJECTIVE /AIM:

The purpose of the study to predict individual's risk for hospital readmission for congestive heart failure using data available at the time of index hospital discharge

MATERIALS AND METHODS

Patients admitted for heart failure previously in madras medical college and Rajiv Gandhi government general hospital and now readmitted with history of heart failure symptoms within the 3 months of discharge are included in the study. Total sample size is 98. The study period is April 2014 – September 2014. . As this study is both prospective and retrospective the lab parameters and clinical parameters of patients previously admitted are obtained from medical records department, Rajiv Gandhi government general hospital.

RESULTS

In our study showed 60% of males and 40% females in medical wards. 40-55 age group showed 44.8% of readmission incidence, 5-9 DAYS of previous admission stay showed 45.91% of readmissions. 35.7% of patient had coronary artery disease as underlying cause. 26.5% had diabetes mellitus as most common comorbid illness. 15.3% has lack of compliance as precipitating factor.

CONCLUSION

The inferences attained from the study are

1. Readmissions are common in the younger age group patients
2. Underlying causes are correctable by surgical measures
3. Use of drugs that decrease the disease progression not used appropriately as there is lack of compliance and awareness
4. Diabetes mellitus and heart failure commonly are associated in many patients
5. Severe left ventricular systolic dysfunction and diastolic dysfunction is the most common echocardiogram finding associated

KEY WORDS

Heart failure, readmissions, ACE inhibitors, diabetes mellitus

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INTRODUCTION

Heart failure is a worldwide problem and the principal complication of all heart disease. Approximately one-half of the patients have moderately to severely reduced LV systolic function, and 50 to 76 percent of patients admitted with acute heart failure had a prior history of heart failure.

Despite dramatic improvement in outcomes with medical therapy, admission rates following heart failure hospitalization remain high, with ≥50% patients readmitted to hospital within 6 months of discharge. Recurrent heart failure and related cardiovascular conditions account for only about half of readmissions in patients with heart failure, whereas other comorbid conditions account for the rest. Individually, several physiological indices of heart failure severity do anticipate higher event rates. Many correlate strongly with elevated filling pressures, such as jugular venous pressure, orthopnea, and echocardiographic filling patterns.

Given the impact of high readmission rates to hospitals, it is important for individual hospitals to identify which patients may be at highest risk of being readmitted.

Certain risk factors, such as patient age, race, diagnoses, length of stay (LOS), comorbidities, insurance, disposition, and prior hospitalizations, are well-documented. Our study aims at predicting the individual's risk for hospital readmission for congestive heart failure using data available at the time of index hospital discharge.

AIMS AND OBJECTIVES

AIM & OBJECTIVES

The purpose of the study to predict individual's risk for hospital readmission for congestive heart failure using data available at the time of index hospital discharge

PRIMARY OBJECTIVE:

The purpose of the study to predict individual's risk for hospital readmission for congestive heart failure using data available at the time of index hospital discharge

SECONDARY OBJECTIVE(S):

- To study the demographic characteristics of the patients.
- To analyze the patient's treatment pattern and follow-up arrangements.
- To determine the underlying and precipitating causes of their illness and therefore readmission.
- To assess the knowledge, attitude and participatory qualities of the patients and their contribution to the readmission.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

ACUTE HEART FAILURE SYNDROME

It can be defined as the new onset or recurrence of symptoms developing gradually or rapidly, signs requiring urgent or emergent treatment and resulting in hospitalization

EPIDEMIOLOGY

The magnitude of heart failure is difficult to assess with accuracy as the population based study of its incidence, prevalence, and prognosis are lacking[1]. The prevalence of heart failure increases with age. Risk was prospectively estimated to be 62%, 17%, 10%, 8%, 3%, 2% for coronary artery disease, cigarette smoking, hypertension, obesity, diabetes mellitus, and valvular heart disease respectively in US. Rheumatic cause of cardiac disease remains a major cause of heart failure in Asia and Africa. As socioeconomic development going on in developing countries like Asia, the epidemiology of heart failure similar to that of western society

CLASSIFICATION

EUROPEAN SOCIETY OF CARDIOLOGY GIDELINES

1. New onset or de novo heart failure; present for first time with symptoms of heart failure or they may not have prior history or have risk factors for heart failure (HF stage A according to ACC/

AHA guidelines)or pre-existing structural heart disease(heart failure stage B according to ACC/AHAguidelines). Most of them develop AHFS in the setting of acute coronary syndromes.

2. Worsening chronic HF; patients have prior history of chronic heart failure(HF stage C according to the ACC/ AHA guidelines) and with decompensation episode. This is about 80% of patients hospitalized with AHFS. Triggering event may be severe chronic symptoms than by an abrupt change in clinical condition

   Clinical scenarios at the time of presentation

   1. Clinical pulmonary edema
   2. Hypertensive heart failure
   3. Cardiogenic shock
   4. Isolated right sided heart failure
   5. Acute coronary syndrome and heart failure
   6. Worsening or decompensated chronic heart failure

ETIOLOGIES OF HEART FAILURE WITH DECREASED EJECTION FRACTION[2]

Myocardial disease
Coronary artery disease
Myocardial infarction*
Myocardial ischemia*
Chronic pressure overload
Hypertension*
Obstructive valvular disease*
Chronic volume overload
Regurgitant valvular disease
Intracardiac (left-to-right) shunting
Extracardiac shunting
Nonischemic dilated cardiomyopathy
Familial/genetic disorders
Infiltrative disorders*
Toxin/drug-induced damage
Metabolic disorder*
Viral or other infectious agents
Disorders of rate and rhythm
Chronic bradyarrhythmias
Chronic tachyarrhythmias
Pulmonary heart disease
Cor pulmonale
Pulmonary vascular disorders
High-output states
Metabolic disorders
Thyrotoxicosis
Nutritional disorders (beriberi)
Excessive blood flow requirements
Systemic arteriovenous shunting
Chronic anemia

PATHOPHYSIOLOGY

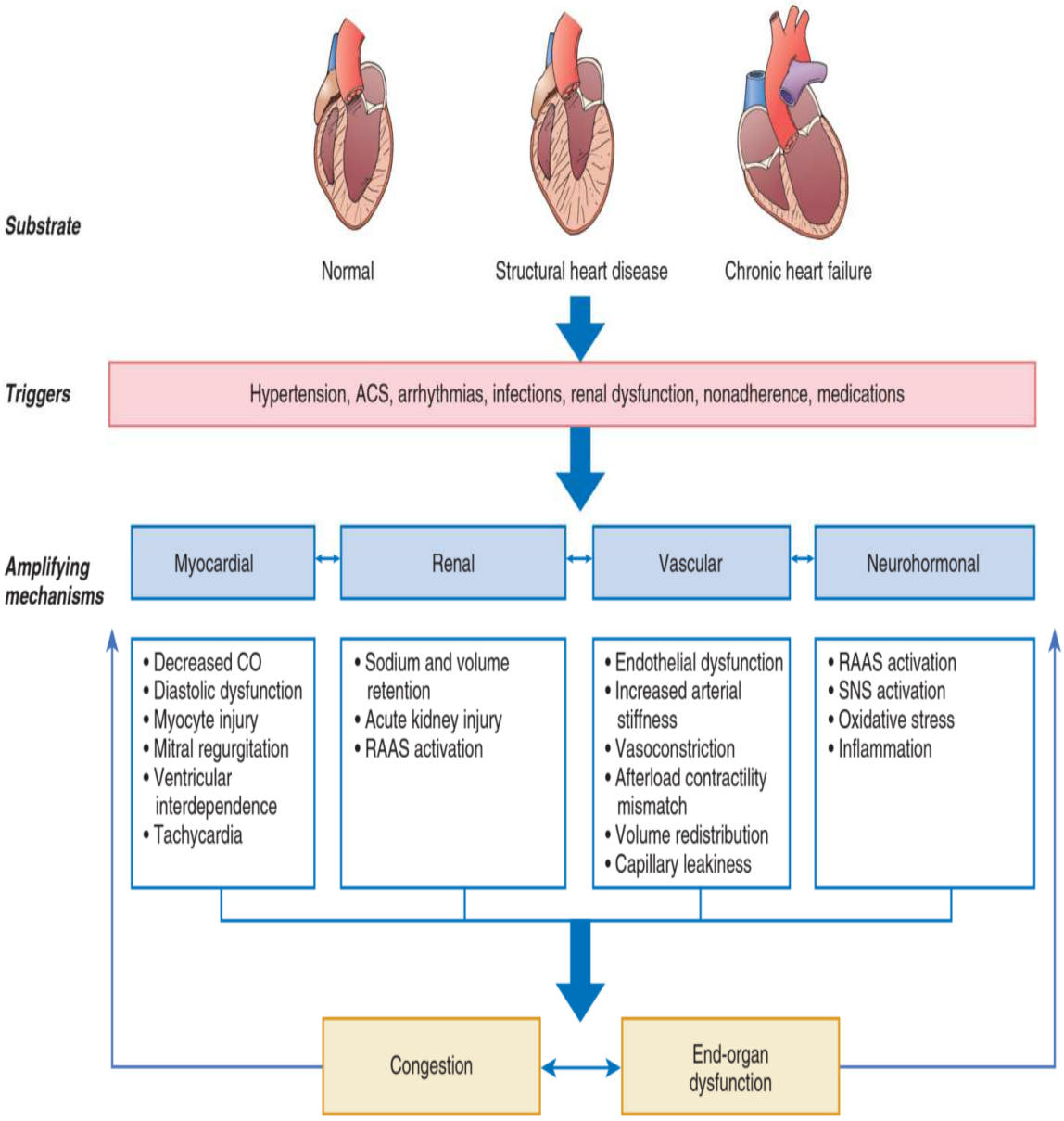
1. NEURO HORMONAL ACTIVATION
2. LEFT VENTRICULAR REMODELLING

   Are the primary determinants in disease progression

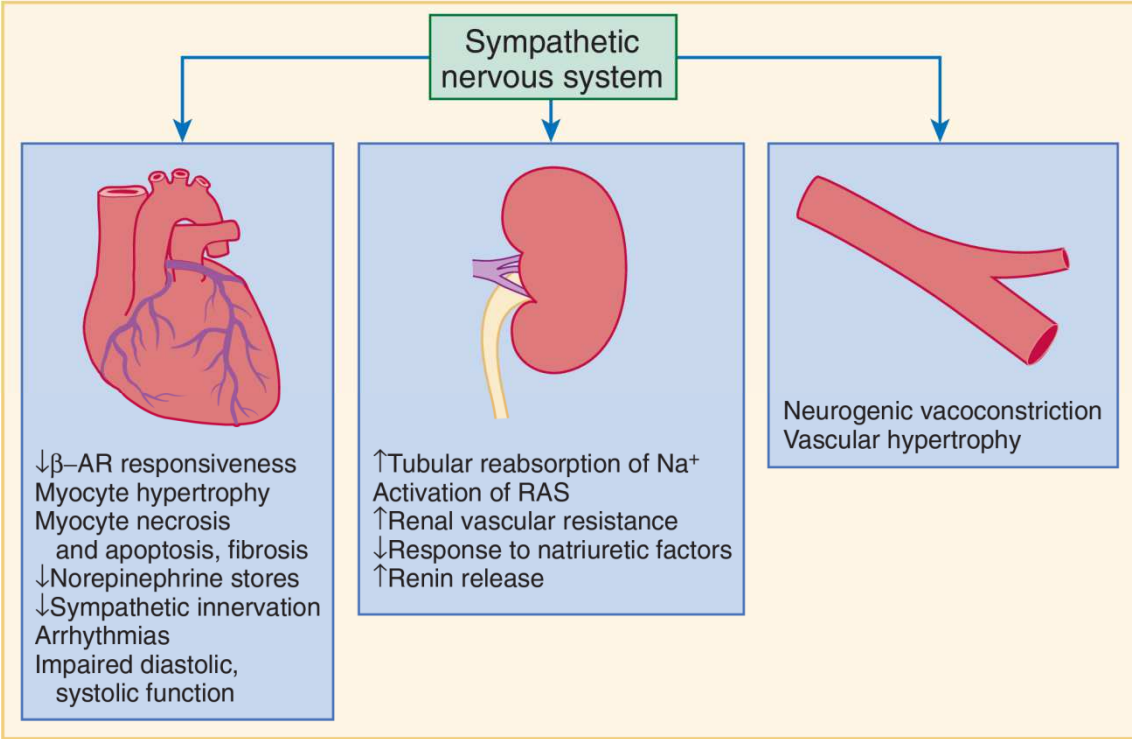
   Heart failure occurs after a primary event like myocarditis which leads to loss of normal force of cardiac contractrility which

damages cardiac muscles. Substrate refers to cardiac structure and function. The underlying substrate may be of normal ventricular function, for example, patients without a prior history of HF who develop AHF because of sudden changes in ventricular function from an insult example myocardial infarction . Alternatively, some patients may have no prior history of HF but abnormal substrate (e.g., stage B patients which is asymptomatic LV dysfunction) with a first presentation of HF (de novo HF). Finally, most patients with AHF have a substrate of chronic compensated HF and then develop decompensation.

SCHEMATIC REPRESENTATION OF PATHOPHYSIOLOGY OF HEART FAILURE

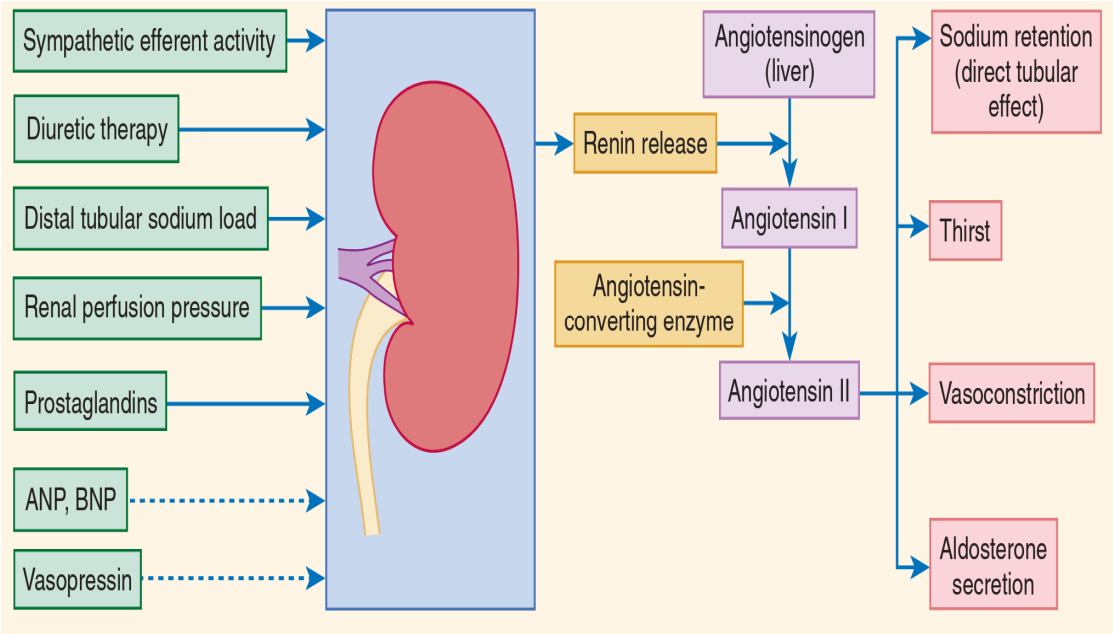


ACTIVATION OF SYMPATHETIC SYSTEM



1. In heart –there is desensitization of beta receptors, myocyte hypertrophy, necrosis , apoptosis and fibrosis
2. Kidney- reactivation of RAS leading to increased salt and water retention and reduce response to natriuretic factors
3. Peripheral vessels- neurogenic vasoconstriction and vascular hypertrophy

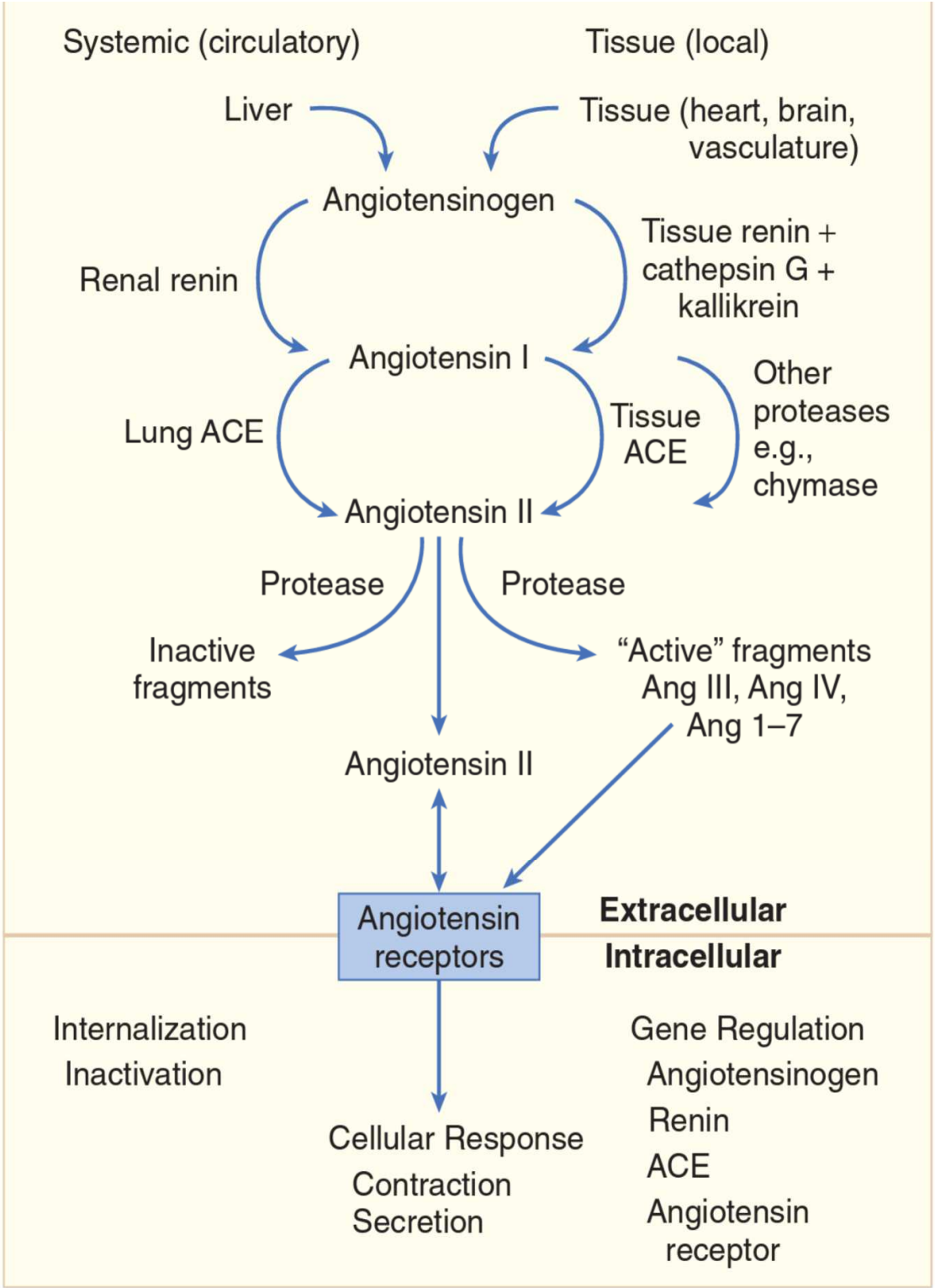
2. ACTIVATION OF THE RENIN ANGIOTENSIN SYSTEM[3]



RAS activation in heat failure occurs comparatively later than the activation of SNS. Mechanism of RAS activation in heart failure occurs as a result of:

1. Decreased renal perfusion
2. sympathetic activity
3. reduced sodium that is filtered reaching macula densa

ANGIOTENSIN SYNTHESIS AND DEGRADATION



Systemic and tissue components of renin-angiotensin system Angiotensin II exerts its effect through two receptors.

1. AT1 receptors activation leads to vasoconstriction, aldosterone secretion, cell growth and catecholamine release. It is predominant in the vascular tissue
2. AT2 , activation of this receptors causes vasodilation, natriuresis, inhibition of cell growth and bradykinin releas. In myocardium AT2 receptors are predominant in 2: 1 molar ratio.
3. In heart failure AT1 down regulated resulting in the ratio of AT1 to AT2 recptors to decrease

ALDOSTERONE SECRETION[`4]

Aldosterone secreted from zona glomerulosa of adrenal cortex by the action of angiotensin II. It promotes the reabsorption of sodium in exchange of potassium in the distal nephron. Aldosterone provokes endothelial cell dysfunction, inhibition of NE uptake, baroreceptor dysfunction, any or all lead to worsening of HF. The action of aldosterone in the cardiovascular system may be due to oxidative stress with resultant inflammation in target tissues, low dose spironolactone increased the survival of patients with systolic heart failure and also improves the

survival after myocardial infarction , independent of changes in electrolyte status or volume

OXIATIVE STRESS[5,6]

Oxidative stress in the heart may be due to the increased production of ROS or reduced antioxidant capacity , which may arise secondary to neurohormonal stimulation (angiotensin II, alpha-adrenergic agonists, ET-1), mechanical strain of the myocardium, or inflammatory cytokines (tumor necrosis factor, IL-1).

NEUROHORMONAL ALTERATION OF RENAL FUNCTION[7,8]

Salt and water retention due to either “forward” failure, due to inadequate renal perfusion as a consequence of impaired cardiac output, or “backward” failure, in which increased venous pressure in favoring transudation of salt and water from the intravascular to the extracellular compartment.

These mechanisms have largely been supplanted by the concept of decreased effective arterial blood volume, which postulates that despite blood volume expansion in HF, inadequate cardiac output sensed by baroreceptors in the vascular tree leads to a series of compensatory neurohormonal adaptations that resemble the homeostatic response to acute blood loss..

Increased renal sympathetic nerve activation leads to

i. Vasconstriction leads to increased renal tubular water, sodium reabsorption throughout the nephron.
ii. Nonosmotic release of arginine vasopressin (AVP)
iii. Endothelin production

ARGININE VASOPRESSIN

(i) AVP is a pituitary hormone that plays a central role in the regulation of free water clearance and plasma osmolality

(ii) AVP is released in response to an increase in plasma osmolality, leading to increased retention of water from the proximal duct.

(iii) In heart failure it is nonosmotic release and contribute to the hyponatremia in HF.

(iv) The cellular effects mediated mainly by three types of receptors, termed V_{1a}, V_{1b}, and V_2. The V_{1a} receptor is present primarily in vascular smooth muscle cells. The V_{1b} mainly in the central nervous system.

(v) The V_2 receptors are found in the epithelial cells in the renal collecting duct and the thick ascending limb.

(vi) AVP receptors are members of the G protein–coupled receptors.

(vii) The V_{1a} receptor mediates vasoconstriction, platelet aggregation, and stimulation of myocardial growth factors;

(viii) the V_{1b} receptor modulates adrenocorticotropic hormone secretion from the anterior pituitary;

(ix) V_2 receptor mediates antidiuretic effects by stimulating adenylyl cyclase to increase the rate of insertion of vesicles containing water channels into the apical membrane.

(x) Preferential action of antagonists V2 receptor causes retention of sodium, increase in plasma renin activity, and plasma AVP, in contrast that of V1 results in the raised cardiac output.

(xi) The vaptans, vasopressin receptor antagonists with V_{1a} (relcovaptan) or V_2 (tolvaptan, lixivaptan) selectivity or nonselective V_{1a}/V_2 activity (conivaptan), have been shown to reduce body weight and to reduce hyponatremia.

The thirst center is stimulated by Angiotensin II and provokes the release of AVP and aldosterone, which can both lead to further dysregulation of salt and water homeostasis.

Natriuretic Peptides [9,10,11]

(i) The natriuretic peptide system consists of five structurally similar peptides, termed ANP, urodilantin (an isoform of ANP), BNP, C-type natriuretic peptide (CNP), and dendroaspis natriuretic peptide (DNP)

(ii) Released in response to increase in atrial or myocardial strech

(iii) ANP in response to raise in atrial pressure is released in short bursts

(iv) BNP regulation is transcriptionaly mediated in response to chronic increases in atrial or ventricular pressure

(v) ANP and BNP are secreted as prohormones cleaved by corin and furin to yield large inactive substrate like N-terminal fragments (NT-ANP or NT-BNP) and smaller biologically active peptides (ANP or BNP).

(vi) ANP has a short half-life of approximately 3 minutes, BNP has a plasma half-life of 20 minutes.

(vii) CNP, located in the vasculature, is also released as a prohormone that is cleaved into a inactive form (NT-CNP) and active form (CNP).

(viii) 1.increases excretion of sodium and water

2.inhibits the release of renin and aldosterone

3.inhibits fibrosis and increasing lusitrophy

(ix) Natriuretic peptides are degraded by neutral endopeptidase. Inhibition of NEP may further potentiate the renal actions of ANP and BNP

(x) Candoxatrilat , an endopeptidase inhibitor parentral formulation leads to reduction of right and left ventricular preload and that is

associated with fall in serum norepinephrine level and decrease in serum vasopressin level.

(xi) Vasopeptidase inhibitors, such as omapatrilat, that inhibit both neutral endopeptidase and ACE were developed with the intent of inhibiting the RAS while concurrently elevating natriuretic peptide levels.

In advanced heart failure ANP and BNP effects blunted due to the low renal perfusion pressure ,molecular form of natriuretic peptide is altered, decreased natriuretic receptors.

EVALUATION OF THE PATIENT WITH HEART FAILURE

Clinical Classification

1. Decompensated heart failure
2. Acute hypertensive heart failure
3. Cardiogenic Shock

Diagnostic Criteria for Heart Failure in Population-Based Studies

FRAMINGHAM CRITERIA		
MAJOR CRITERIA	**MINOR CRITERIA**	**MAJOR OR MINOR CRITERIA**
Paroxysmal nocturnal dyspnea or orthopnea Neck-vein distention Rales Cardiomegaly Acute pulmonary edema S_3 gallop Increased venous pressure, >16 cm H_2O Hepatojugular reflux	Ankle edema Night cough Dyspnea on exertion Hepatomegaly Pleural effusion Vital capacity decreased by one third from maximal capacity Tachycardia (rate > 120 beats /min)	Weight loss > 4.5 kg in 5 days in response to treatment

OTHER CRITERIA FOR THE DIAGNOSIS OF HEART FAILURE

TABLE 2. **Vasan and Levy's Criteria for the Diagnosis of Diastolic Heart Failure**[12]

Definitive diagnosis
Definitive clinical evidence of heart failure, and
Normal left ventricular systolic function with ejection fraction >50% determined in the 72 hours following clinical decompensation and
Objective evidence of diastolic dysfunction in the hemodynamic study (increase in diastolic pressure with normal or reduced diastolic volume)
Probable diagnosis
Definitive clinical evidence of heart failure, and
Normal left ventricular systolic function with ejection fraction >50% determined in the 72 hours following clinical decompensation
Possible diagnosis
Definitive clinical evidence of heart failure, and
Normal left ventricular systolic function with ejection fraction >50% determined outside of the 72 hours following clinical decompensation

DIAGNOSTIC TESTING

Measurement of Blood Chemistry and Hematologic Variables

Blood chemistries and hematologic variables are routinely measured as part of the initial heart failure evaluation. Hyponatremia is the most common electrolyte abnormality in heart failure patients, particularly during periods of decompensation. Although hyponatremia is associated with significantly higher in-hospital and follow-up mortality and longer hospital stays. Hyponatremia, however, has been associated with impaired cognitive and neuromuscular function. Besides increasing the risk of cardiac arrhythmias, it can cause further muscle weakness. Additional electrolyte abnormalities include hypomagnesemia and hypophosphatemia.

Renal function should be measured as part of the initial evaluation. Dronedarone, an agent used to treat intermittent or recurrent atrial fibrillation, can also increase creatinine levels. [14] Impaired renal function at baseline and worsening during hospitalization are both potent predictors of poor outcome.

Diabetes is common in heart failure patients. Because diuretics can cause gout, measurement of uric acid levels can help in management of the patient. Abnormalities in aspartate aminotransferase and alanine aminotransferase may occur in heart failure patients as a consequence of either hemodynamic derangements or medications, and it is important to

follow levels periodically. An unexpected increase in prothrombin time in patients receiving warfarin therapy may be an early harbinger of decompensation as it may reflect impaired synthetic capacity of a congested liver. Albumin levels are an indication of the patient's nutritional status, and they may be depressed because of poor appetite or impaired absorption across an engorged bowel wall. Elevations in bilirubin are most often seen during episodes of severe decompensation.

Low hemoglobin levels have been associated with more severe symptoms, reduced exercise capacity and quality of life, and increased mortality[15,16] . Although anemia may be a consequence of chronic disease in heart failure patients, a low hemoglobin level should trigger an evaluation to detect treatable causes. The leucocyte count and differential are helpful in detecting the presence of infection that is responsible for destabilizing a previously well-compensated patient and could provide a clue that heart failure is due. A BNP level above 400 pg/mL in this setting make the diagnosis of heart failure likely. A level of NT-proBNP of 300 pg/mL has been set as a cut off for excluding or including the diagnosis of heart failure

Chest Radiography

It is taken to rule out other causes of dyspnoea. A “butterfly” pattern of alveolar opacities that fan out bilaterally from engorged hilar pulmonary arteries to the periphery of the lungs is the classic pattern of

congestion seen in decompensated heart failure . Pleural effusions or fluid in the right minor fissure may also be seen. The chest radiograph may also provide an indication that heart failure is due to coronary, valvular, or pericardial disease when calcification is noted in the appropriate position.

Electrocardiography

Sinus tachycardia secondary to sympathetic nervous system activation is seen with advanced HF or during episodes of acute decompensation. Atrial arrhythmia on the ECG, as well as the ventricular response, may provide clues to the cause of HF and gives the reason for decompensation. Increased QRS voltage may suggest left ventricular hypertrophy, if right ventricular hypertrophy is present primary or secondary pulmonary hypertension should be considered. Low QRS voltage suggests the presence of an infiltrative disease or pericardial effusion. The presence of Q waves suggests that HF may be due to ischemic heart disease; new or reversible ST changes identify acute coronary ischemia, which may be present even when chest pain is absent. acute coronary ischemia is a leading cause of acutely decompensated HF Prolongation of the PR interval is common in patients in this setting and may be due to intrinsic conduction disease but also may be seen in patients with infiltrative cardiomyopathy. With the advent of cardiac

resynchronization therapy, evaluation of the QRS complex has become a critical part of the clinical assessment in that it provides important information regarding the causes of HF, as well as providing pivotal information regarding the therapeutic approach. The QT interval often is prolonged in patients with HF, which may be due to electrolyte abnormalities, myocardial disease, or effects of commonly used drugs, such as antiarrhythmics.

Echocardiogram

Transthoracic echocardiography is an important part of the evaluation of HF. It is particularly well suited for evaluating the structure and function of both the myocardium and heart valves and providing information about intracardiac pressures and flows. For patients with HFpEF, left ventricular volumes and systolic function can be assessed semiquantitatively, or quantified using the biplane method and the modified Simpson's rule. Diastolic function is assessed using Doppler measurements, including analyses of the mitral valve inflow pattern (early [E] and atrial [A] waveforms), tissue velocities at the mitral valve annulus, pulmonary vein flow, and the left atrial volume indexed to body surface area. Diastolic dysfunction can be further classified as grades I to III based on the foregoing measurements, with incremental prognostic importance in HF as worsening grades of diastolic dysfunction are noted.

Pulmonary hypertension in patients without significant systolic dysfunction or pulmonary disease suggests that diastolic dysfunction may be present.

Another advantage of echocardiography is the ability to noninvasively estimate right-sided heart pressures. For example, right atrial pressures are estimated by the inferior vena cava (IVC) diameter and the relative change in diameter on inspiration. Normal IVC diameter and inspiratory collapse of at least 50% are associated with normal right atrial pressures, whereas increased IVC diameter and smaller inspiratory changes indicate elevated right atrial pressure MRI and CADIAC CT can also be done, MRI predicts morphology, function, tissue metabolism but CT can do evaluate only morphology and function

TREATMENT OF HEART FAILURE

NON –PHARMACOLOGICAL THERAPIES

DIET

1. Dietary restriction of sodium 2-3 gram daily is recommended for all patients, regardless of left ventricular ejection fraction.
2. Fluid restriction less than 2 litre daily if serum sodium less than 130mEq /L
3. Prealbumin and caloric evaluation

OTHER THERAPIES

1. Continuous positive airway pressure if diagnosis of sleep apnoea is present, symptoms of dyspnoea improves
2. Oxygen supplementation
3. Depression if present treated with appropriate therapy

ROUTINE HEALTHCARE

1. Avoidance of alcohol less than 2 drink in male and 1 drink in female. In alcoholic cardiomyopathy, total abstinence is recommended
2. Vaccinations like pneumococcal and influenza is recommended
3. Exercise training , moderate exercise activity for 30 minutes at least 5 days per week

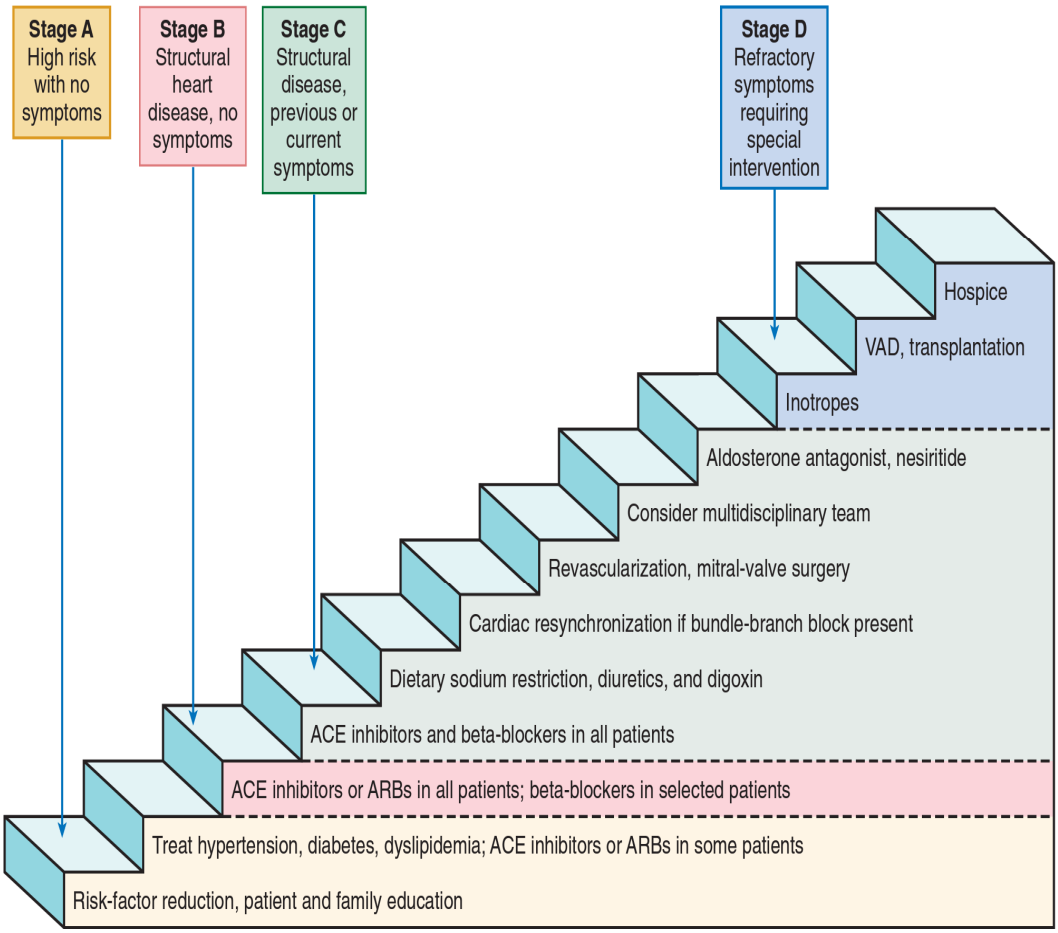
PHARMACOLOGICAL THERAPIES

It is based on the stages of heart failure

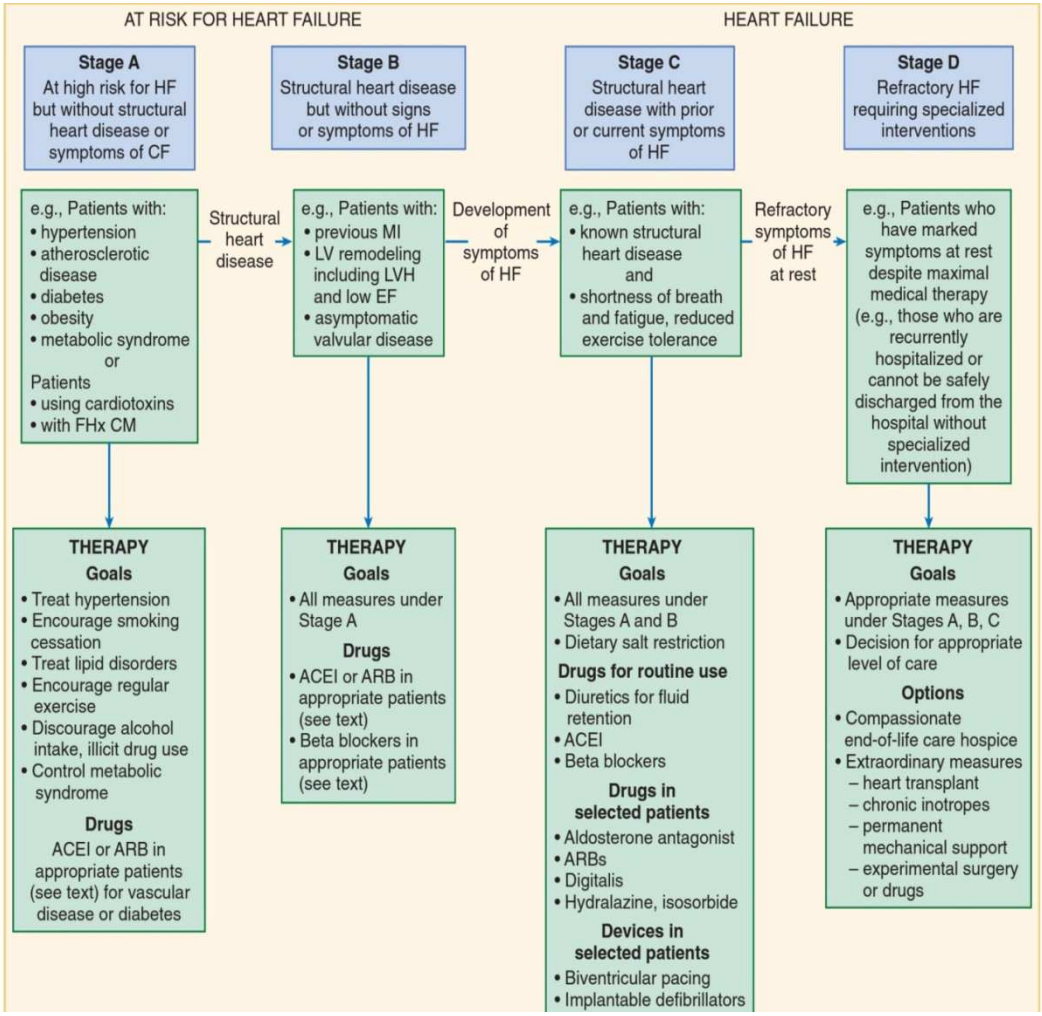
STAGES OF HF[23]

ACC/AHA STAGES	
A	At high risk for HF but without structural heart disease or HF symptoms
B	Structural heart disease but without signs or symptoms of HF
C	Structural heart disease with previous or current symptoms of HF
D	Refractory HF requiring specialized interventions

AHA guideline for treatment of heart failure



Goal of treatment for each stages of heart failure



LOOP DIURETICS [19,20]

DRUG	INITIAL DAILY DOSE(S)	MAXIMUM TOTAL DAILY DOSE	DURATION OF ACTION
Loop Diuretics*			
Bumetanide	0.5-1.0 mg once or twice	10 mg	4-6 hours
Furosemide	20-40 mg once or twice	600 mg	6-8 hours
Torsemide	10-20 mg once	200 mg	12-16 hours
Ethacrynic acid	25-50 mg once or twice	200 mg	6 hours
Thiazide Diuretics†			
Chlorthiazide	250-500 mg once or twice	1000 mg	6-12 hours
Chlorthalidone	12.5-25 mg once	100 mg	24-72 hours
Hydrochlorthiazide	25 mg once or twice	200 mg	6-12 hours
Indapamide	2.5 mg once	5 mg	36 hours
Metolazone	2.5-5.0 mg once	20 mg	12-24 hours
Potassium-Sparing Diuretics			
Amiloride	12.5-25 mg once	20 mg	24 hours
Triamterene	50-75 mg twice	200 mg	7-9 hours
AVP Antagonists			
Satavaptan	25 mg once	50 mg	NS
Tolvaptan	15 mg once	60 mg	NS
Lixivaptan	125 mg once	250 mg	NS
Conivaptan (IV)	20 mg IV loading dose, followed by 20 mg/day continuous IV infusion	40 mg IV infusion/day	7-9 hours
Sequential Nephron Blockade			
Metolazone	2.5-10 mg once PLUS loop diuretic		
Hydrochlorothiazide	25-100 mg once or twice PLUS loop diuretic		
Chlorothiazide (IV)	500-1000 mg once PLUS loop diuretic		

Furosemide, bumetanide, and torsemide, are the loop diuretics act by reversibly inhibiting the Na+-K+-2Cl− symporter.

Mechanisms of Action

By inhibiting the concentration of solute within the medullary interstitium, these drugs also reduce the driving force for water resorption in the collecting duct, even in the presence of AVP. Furosemide acts as a venodilator and reduces right atrial and pulmonary capillary wedge pressure within minutes when given intravenously (0.5 to 1.0 mg/kg).

Thiazide and Thiazide-Like Diuretics

The benzothiadiazides, also known as thiazide diuretics, were the initial class of drugs synthesized to block the Na+-Cl− transporter in the distal nephron. As a result of its action the sodium delivery is increased in the collecting tubules and that leads to enhanced sodium reabsorption in this segment in exchange of H+ and K+ ions that leads to hypokalemia

Mechanisms of Action

The efficacy of thiazide diuretics is dependent, at least in part, on proximal tubular secretion to deliver these agents to their site of action..

Mineralocorticoid Receptor Antagonists

These agents are used in patients more for their ability to antagonize the renin angiotensin aldosterone system than for their diuretic properties.

Potassium-Sparing Diuretics

Triamterene and amiloride are referred to as potassium-sparing diuretics. Its action is carried out by the blockade of epithelial sodium channel present in the principal cells of late distal tubule and collecting duct

Carbonic Anhydrase Inhibitors

The use of these agents in patients with HF is restricted only in occasions of metabolic alkalosis that occurs at times of ECF contraction in diuretic usage.

Diuretics started at lower level and dose is increased until patient attains the euvolemic state. . Intravenous administration of loop diuretics helps to decrease the symptoms of acute heart failure symptoms briefly. On attaining the steady state of euvolemia , small doses of diuretics are given repeatedly like thrice a day orally, than a single large dose

Complications of Diuretic Use

Electrolyte and Metabolic Disturbances

It can lead to potassium depletion, which can predispose the patient to significant cardiac arrhythmia.

The serum potassium should be within the range of 4 to 5 milliequivalent/L

- Hyponatremia
- Hypomagnesemia
- Metabolic alkalosis
- Hhyperglycemia
- Hhyperlipidemia
- Hhyperuricemia

Potassium chloride supplementation or acetazolamide used to relieve metabolic alkalosis

- Hypotension and Azotemia
- Neurohormonal Activation
- Ototoxicity, which is more frequent with ethacrynic acid than the other loop diuretics, reversible. Ototoxicity occurs most frequently with rapid intravenous injections, and least frequently with oral administration.
- Diuretic Resistance.

Drugs for the Prevention and Treatment of Chronic Heart Failure[13,14]

AGENT	INITIATING DAILY DOSE	MAXIMAL DAILY DOSE
Angiotensin-Converting Enzyme Inhibitors		
Captopril	6.25 mg 3×	50 mg 3×
Enalapril	2.5 mg twice	10 mg twice
Lisinopril	2.5-5.0 mg once	20 mg once
Ramipril	1.25-2.5 mg once	10 mg once
Fosinopril	5-10 mg once	40 mg once
Quinapril	5 mg twice	40 mg twice
Trandolapril	0.5 mg once	4 mg once
Angiotensin Receptor Blockers		
Valsartan	40 mg twice	160 mg twice
Candesartan	4-8 mg once	32 mg once
Losartan	12.5-25 mg once	50 mg once
Beta Blockers		
Carvedilol	3.125 mg twice	25 mg twice (50 mg twice in patients weighing > 85 kg)
Carvedilol-CR	10 mg once	80 mg once
Bisoprolol	1.25 mg once	10 mg once
Metoprolol succinate CR	12.5-25 mg qd	200 mg once
Aldosterone Antagonists		
Spironolactone	12.5-25 mg once	25-50 mg once
Eplerenone	25 mg once	50 mg once
Other Agents		
Combination of hydralazine–isosorbide dinitrate	10-25 mg/10 mg 3×	75 mg/40 mg 3×
Fixed dose of hydralazine–isosorbide dinitrate	37.5 mg/20mg (one tablet) 3×	75 mg/40 mg (two tablets) 3×
Digoxin*	0.125 mg qd	≤0.375 mg/day†
Ivabradine	5 mg twice daily	7.5 mg twice daily‡

N-3 Polyunsaturated (Omega-3) Fatty Acids

These drugs have favorable effects on inflammation, including a reduction of endothelial activation and production of inflammatory cytokines, platelet aggregation, autonomic tone, blood pressure, heart rate, and LV function

Ivabradine

Ivabradine acts by decreasing the heart rate. If("funny") current is blocked by this agent that decreases the rate of depolarization during the diastole. It acts mainly in sinoatrial node. So this drug used in patients in sinus rhythm.

It acts from the intracellular site, so it can block the channel while it is open. It is more effective while treating a patient in the fast heart rate, as it's action depends on the frequency of opening of the If channel

Systolic Heart Failure Treatment with the If Inhibitor Ivabradine Trial (SHIFT), in this study patients with severe left ventricular dysfunction proved on echocardiogram is enrolled, the heart rate cut off is 70 beats per minute and above , the dose of the drug is 7.5 mg twice a day. There was a decrease in the hospitalization of about 18% and mortality is also decreased.

BEAUTIFUL trial more than 10000 patients were enrolled, in that patient ejection fraction is fixed at 40% or less and the underlying causes

were post myocardial infarction, hypertensive heart disease, etc. this trial showed that the patient tolerated this drug better as the side effects were low.

Cardiac Glycosides

Digoxin and digitoxin are more commonly used drug in severe LV dysfunction. Digoxin acts by inhibiting the Na^{+}-K^{+}ATPase(sodium potassium adenosine trisphosphate). This leads to an increase in intracellular calcium and hence increased cardiac contractility, because of its ionotropic property.

In the heart failure it acts also by improving the vagal sensitization and therefore counterbalancing the effect of sympathetic overactivity. Its action on renal tubules by blunting the absorption of sodium as the channel is same as that of myocardium.

Dose is started with 0.125 to 0.25 mg daily and the serum digoxin level should be below 1.0 ng/mL in

1. Low BMI patients
2. Old age

Complications of Digoxin Use

Adverse effects of digoxin are

(i) Cardiac arrhythmias including heart block (especially in the elderly) and ectopic and reentrant cardiac rhythms

(ii) Neurologic complaints such as visual disturbances, disorientation, and confusion;

(iii) Gastrointestinal symptoms such as anorexia, nausea, and vomiting serum K+ are monitored, The concomitant use of quinidine, verapamil, spironolactone, flecainide, propafenone, and amiodarone increases the drug level so frequent monitoring to be done.

Drug that affects the serum level of digoxin

DRUG	EFFECT ON SERUM LEVEL	MECHANISM
Amiodarone	Increases	??Renal clearance
Verapamil	Increases	?Renal clearance
Nifedipine	Increases	?Renal clearance
Diltiazem	Increases	?Renal clearance
Quinidine	Increases	Displacement of protein binding, ?renal clearance
Propafenone	Increases	?Renal clearance
Captopril	? Increases	? Renal clearance
Carvedilol	Increases	?Oral bioavailability
Spironolactone	Increases	?Renal clearance
Amiloride	Increases	?Renal clearance
Triamterene	Increases	?Renal clearance
Salbutamol	Decreases	Unknown
Macrolide antibiotics	Increases	Altered gut flora, ?renal clearance
Tetracycline	Increases	Altered gut flora
Indomethacin	Increases	?Renal clearance
Alprazolam	Increases	??Renal clearance
Itraconazole	Increases	?Renal clearance
Rifampin	Decreases	Induction of gut P-glycoprotein
Sucralfate	Decreases	Decreased gut absorption
Cholestyramine	Decreases	Decreased gut absorption
Cyclosporine	Increases	?Renal clearance
St. John's wort	Increases	?Renal clearance

Renin Inhibitors

Aliskiren is the drug in this group which is orally active and it directly inhibits the renin.

Its action similar to that of the ACE inhibitors. ACE inhibitor use leads to raise in renin level in the plasma. This raised renin level can activate renin angiotensin system and decrease the effect of ACE inhibitor. Renin inhibitors used in these situation

Management of Atherosclerotic Disease

Aspirin can be added to prevent any complications of atherosclerosis, studies shown its relevance but not yet proven in heart failure.

Anticoagulation and Antiplatelet Therapy [15]

Treatment with these drugs are recommended in

1. arrhythmias
2. anterior wall myocardial infarction
3. myocardial infarction with clot noted in echocardiogram
4. history of systemic embolization like transient ischemic attack or pulmonary embolism

- treatment continued for three months unless it is contraindicated
- INR maintained at 2 to 3

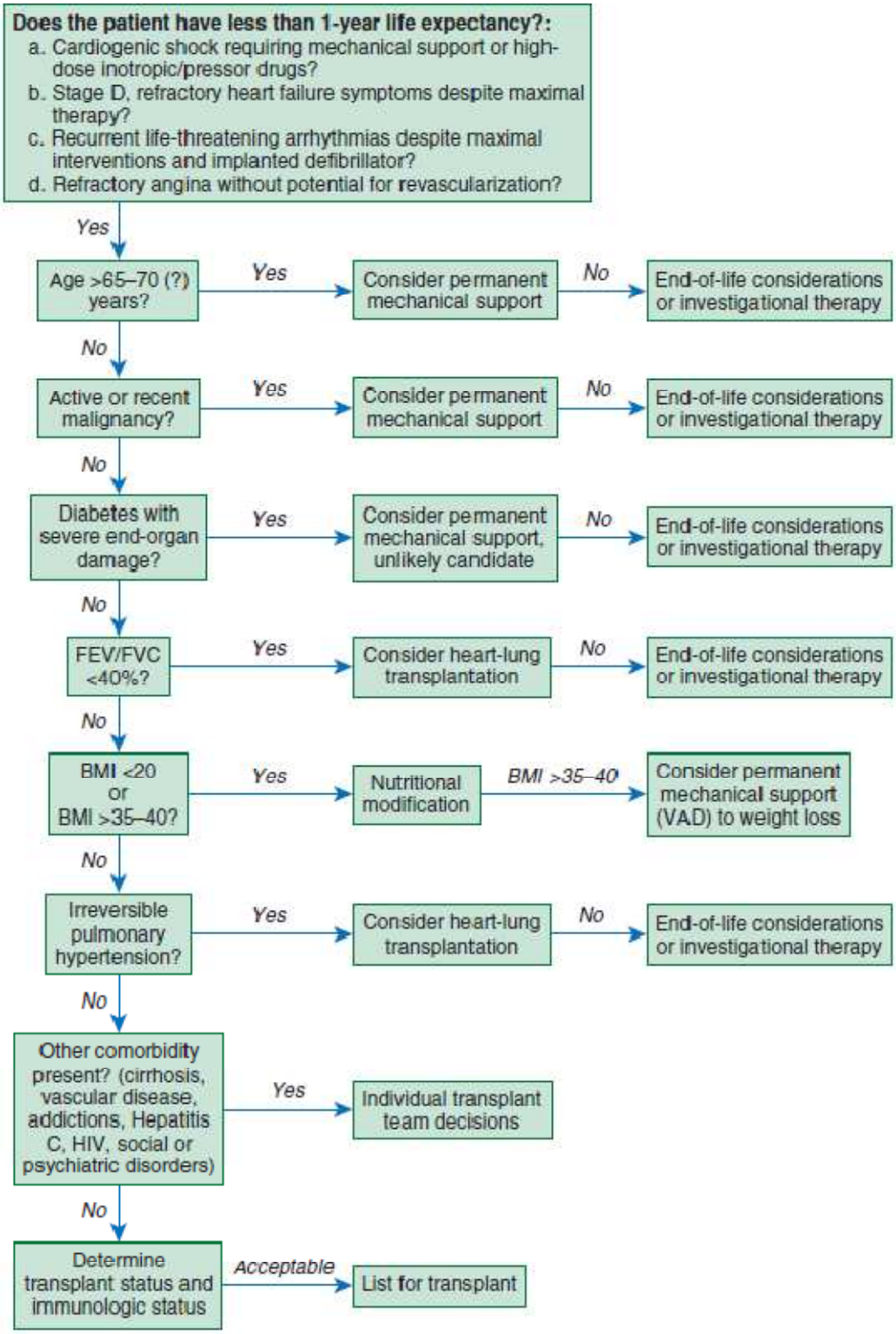
Treatment of Cardiac Arrhythmias

1. atrial fibrillation occurs in 15% to 30% of patients with HF, and is a frequent cause of cardiac decompensation
2. only amiodarone and dofetilide are used as only these drugs does not have negative ionotropism and does not provoke arrhythmia.
3. Drug interactions of amiodarone is high, as it interacts with many drugs that used in heart failure like warfarin and digoxin
4. Dose of amiodarone is decreased to50% in these patients, to avoid drug interactions. As other side effects of amiodarone like hyperthyroidism,hepatitis, pulmonary fibrosis is low in this low dosage about 100 to 200mg per day
5. Dronedarone is a novel antiarrhythmic drug that reduces the incidence of atrial fibrillation and atrial flutter and has electrophysiologic effects similar to those of amiodarone but side effects are less than amiodarone due to absence of iodine moiety.

Device Therapy[16]

Device therapy can improve the quality of life in patients with sinus rhythm. There is decrease in probability of hospitalization in those patients with device therapy

SURGICAL MANAGEMENT

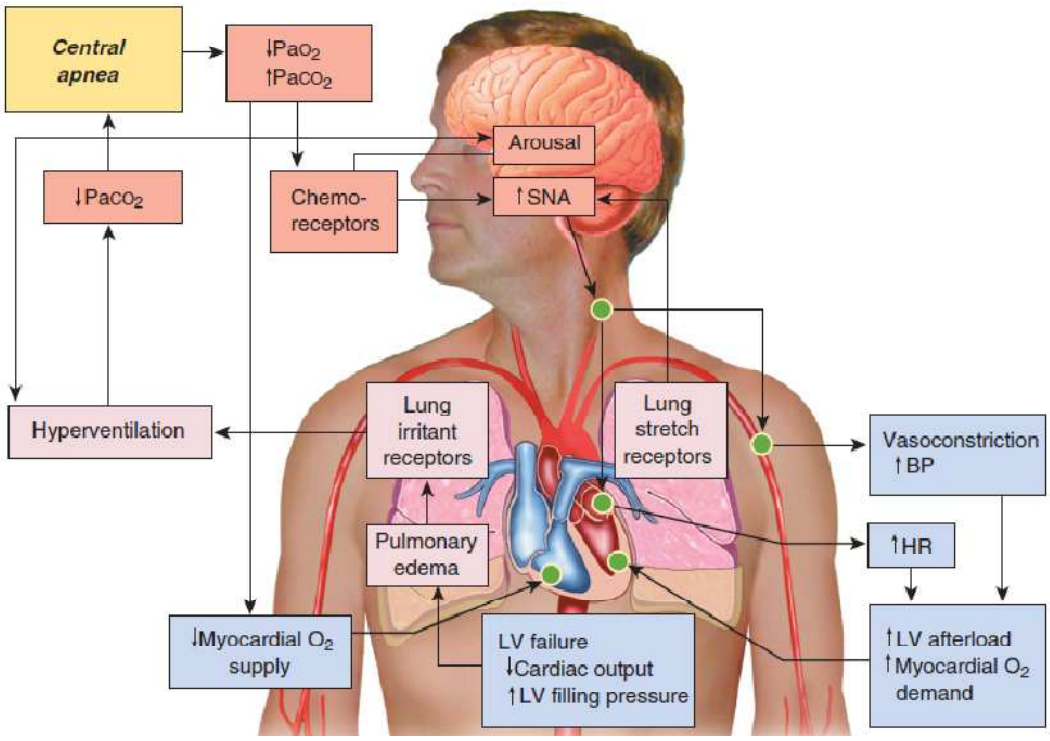


MANAGEMENT OF CSA IN HEART FAILURE[17]

HF patients with a reduced EF (<40%) commonly exhibit sleep-disordered breathing; approximately 40% of patients exhibit central sleep apnea (CSA), commonly referred to as Cheyne-Stokes breathing whereas another 10% exhibit obstructive sleep apnea (OSA)

Risk factors for the development of CSA in HF patients include male gender, age older than 60 years, presence of atrial fibrillation, and hypocapnia. The main clinical significance of CSA in HF is its association with increased mortality.

The potential mechanism(s) for adverse outcomes in HF patients with CSA may be attributed to marked neurohumoral activation, especially norepinephrine.



Continuous positive airway pressure (CPAP) breathing has been shown to improve LV structure and function in patients with OSA or CSA disturbed breathing syndrome.

Disease Management

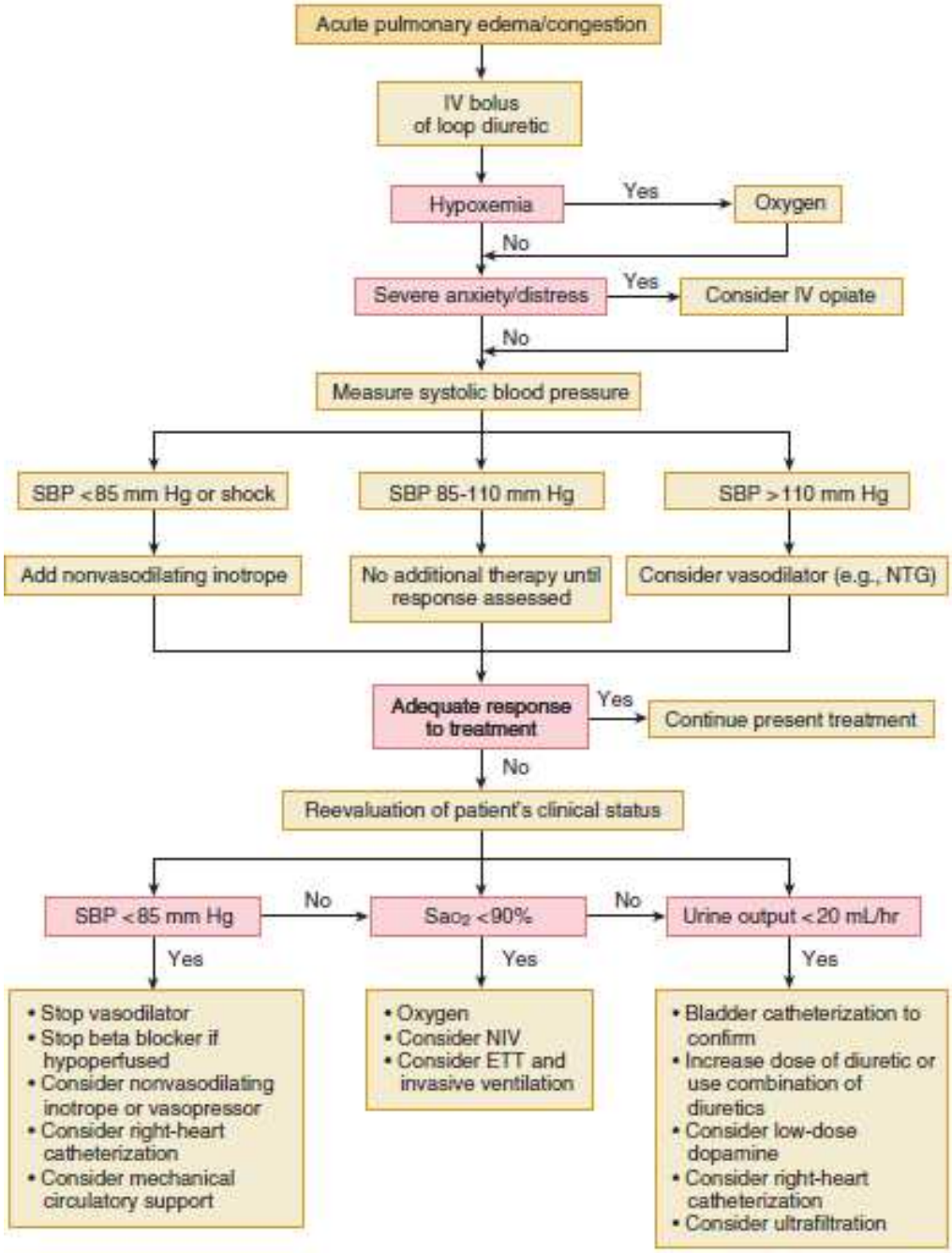
Technology-driven strategies that use low-cost telemonitoring also appear promising in terms of improving HF management and outcomes, emphasizing the importance of team management in the care of these complex patients[18]. Ideal guideline recommended treatment group in a study showed decrease in rehospitalization.

Patients with Refractory End-Stage Heart Failure (Stage D)

Those patients in this stage is referred for advanced treatment like mechanical circulatory support, continuous intravenous positive inotropic therapy, Cardiac transplantation.

PHASES OF MANAGEMENT [21,22]

PHASE I EMERGENT CARE



Phase II: Hospital Care

The main aspect of treatment at the time of hospital admission is the completion of diagnostic workup and the emergent therapeutic process that was started at the time of admission for pulmonary edema and to correct the hemodynamic derangements. The goal is to attain a state of optimal treatment that correct the volume status, symptoms and plan the therapy of chronic heart failure

These are optimized to decrease the duration of stay in the hospital, and intensive care. Surveillance of day to day weight of the patient, input output chart of the patient,orthostatic fall in blood pressure , signs and symptoms of congestion is important during this phase. Echocardiogram and electrocardiogram is done to evaluate the cause of decompensation, as myocardial infarction is frequently associated.

Restriction of sodium less than two gram per day and amount of water or other fluid to less than two litre per day is instituted.

As there is risk of systemic and pulmonary embolization in these patients, to start on warfarin or heparin is indicated unless it is contraindicated as the patient is mostly bed ridden

During hospitalization itself the patient is started on the drugs that is prescribed during the discharge and the most important aspect is to include angiotensin converting enzyme inhibitor and aldosterone antagonist unless the patient urea and creatinine shows a raising trend.

Those who were started on betablocker in a trial showed decreased incidence of ventricular tachycardia, decreased length of hospital stay, decreased mortality compared to the control who were not on the betablocker.

Beta blocker therapy is given unless the patients in cardiogenic shock or persistent hypoperfusion . ACE inhibitors, angiotensin receptor blockers, mineralocorticoid receptor antagonists, and isosorbide dinitrate/hydralazine should be continued unless there is situations of withdrawal.

During this phase patient is taught about the importance of the treatment schedule and the self care Programme arranged to learn about the specific drugs and importance of daily weight measurement and the awareness about the heart failure and behavior therapy is also instituted. self-adjustment of diuretics, exercise programs, and nutritional counseling are also done. Comorbid conditions also given importance to these patients, and advice about its management and care is given.

The Cardiorenal Syndrome in Hospitalized Patients

It is diagnosed, in the presence of the systemic congestion if the patient shows raise in creatinine 0.3 mg/dL or if there is fall in glomerular filtration rate about 25% from the base line.

It is associated with the increase in duration of the stay and postdischarge mortality and morbidity, therefore readmissions Angiotensin converting enzyme inhibitor and aldosterone antagonist are to be discontinued in the presence of increasing trend of creatinine value (BUN value more than 80 mg/dL and creatinine above 3 mg/d)..

In these situations vasodilator therapy like (nitroglycerin or nitroprusside) and (hydralizine or isosorbide dinitrate) is used to relieve the congestion.

These situations leads to a state of diuretic resistance, so there is a need of raising dose of diuretics. Ultrafiltration can be used to relieve the symptoms but vasodilators is superior as they preserve the renal function.

Phase III: Predischarge Planning

It is based mainly on giving the optimial therapy to each patient, patient must be stabilized on the chronic outpatient therapy and the side effects of the drugs must be minimized by prescribing dosage that are appropriate

Criteria for discharging the patient

Recommended for All Patients with Heart Failure (HF)
• Exacerbating factors addressed • Near optimal volume status observed • Transition from intravenous to oral diuretic successfully completed • Patient and family education completed, including clear discharge instructions • LVEF documented • Smoking cessation counseling initiated • Near optimal pharmacologic therapy achieved, including ACE inhibitor and beta blocker (for patients with reduced LVEF), or intolerance documented • Follow-up clinic visit scheduled, usually for 7-10 days later
Interventions to Be Considered for Patients with Advanced HF or Recurrent Admissions for HF
• Oral medication regimen stable for 24 hours • No intravenous vasodilator or inotropic agent for 24 hours • Ambulation before discharge to assess functional capacity after therapy • Plans for postdischarge management (scale present in home, visiting nurse or telephone follow-up generally no longer than 3 days after discharge) • Referral for disease management, if available

Phase IV: Postdischarge Management

After the discharge patient must be advised to attend the follow up care within 7 to 10 days. This can be adjusted as earlier as possible in patients with high risk. As the neuohormonal activation due to congestion is the most common reason for recurrence of the symptoms.

The Rehospitalization Problem

In the EVEREST study, followed up the patients with readmissions, showed that 46% were for heart failure, 15% for other cardiovascular causes, and 39% were for noncardiovascular causes. Hospitalizations in heart failure are the major burden in all over the world.

Use of appropriate drugs and dosage that reduce hospitalization are ACE inhibitors and beta blockers that are proven in many studies.

Potential Precipitating Factors of Acute Decompensation in Patients with Chronic Heart Failure

Dietary indiscretion
Inappropriate reduction in HF medications
Myocardial ischemia, infarction
Arrhythmias (tachycardia, bradycardia)
Infection
Anemia
Initiation of medications that worsen the symptoms of HF
Calcium antagonists (verapamil, diltiazem)
Beta blockers

Nonsteroidal anti-inflammatory drugs

Thiazolidinediones

Antiarrhythmic agents (all Class I agents, sotalol [Class III])

Anti-TNF antibodies

Alcohol consumption

Pregnancy

Worsening hypertension

Acute valvular insufficiency

OVERVIEW OF TREATMENT OPTIONS IN HEART FAILURE PATIENTS

INDICATION	ACE INHIBITOR	ARB	DIURETIC	BETA BLOCKER	ALDOSTERONE ANTAGONIST	CARDIAC GLYCOSIDES	CRT	ICD
Asymptomatic LV dysfunction (NYHA class I)	Indicated	If patient is ACE-intolerant	Not indicated	Post-MI Indicated*	Recent MI	(1) For rate control with atrial fibrillation or (2) when improved from more severe HF and in sinus rhythm	May be considered*	Indicated
Symptomatic HF (NYHA class II)	Indicated	Indicated with or without ACE inhibitor	Indicated if fluid retention present	Indicated	Indicated		Indicated†	
Worsening HF (NYHA class III-IV)	Indicated	Indicated with or without ACE inhibitor	Indicated, combination of diuretics	Indicated (under specialist care)	Indicated	Indicated	Indicated‡	Indicated
End-stage HF (NYHA class IV)	Indicated	Indicated with or without ACE inhibitor	Indicated, combination of diuretics	Indicated (under specialist care)	Indicated	Indicated	Indicated‡	Not indicated§

FACTORS THAT ARE CONSIDERED AS PROGNOSTIC MARKERS ARE ILLUSTRATED

HOSPITALIZATION[24]

Poor prognostic factor is need for hospitalization. Mortality rate is high after hospitalization, more within one month of discharge. Contributing factor is poor compliance in many patients who is requiring readmissions.

SURVIVAL IN HEART FAILURE[25]

DURING HOPITALIZATION

Mortality is high if in-hospital stay is decreased. The decrease in duration of hospital stay is associated with increase in post discharge mortality, as the patients are discharged in unstable conditions and thereby readmission rate is increased

LONG TERM MORTALITY[26,27,28]

It is studied in various studies and it states that it is declining. In a study of patients with systolic dysfunction on angiotensin converting enzyme inhibitor along with some taking spironolactone , cardiovascular death incidence or symotomatic failure was 39 % over 3 years.

PREDICTIVE MODELS[29]

Variety of predictors has been identified

HEART FAILURE SURVIVAL SCORE[30] is based on

1. Ejection fraction
2. Peak o2
3. Serum sodium
4. Mean blood pressure
5. Resting heart rate
6. Coronary vascular disease
7. ECG showing intraventricular conduction delay

SEATLE HEART FAILURE MODEL is a multivariable analysis model using demographic and clinical markers to predict the survival.

Effect of age - Increasing age has impact over mortality and morbidity[31]

Effect of gender[32]

Women having better prognosis than men, in PRAISE, PRAISE II, MERIT HF, VEST, PROMISE trial proposed this.

CAUSE OF CARDIOMYOPATHY[33]

Peripartum cardiomyopathy has better prognosis. Infiltrative, HIV, DOXORUBICIN therapy, ischemic heart disease, connective tissue disease has worse prognosis.

But prognosis is same in hypertension, substance abuse, myocarditis.

SOLVD treatment trial showed diabetes increases the mortality in all cause HF.

SEASONAL VARIATION[34,35]

Winter spring predominance in hospitalization, fatal events are more common in winter in a large study from france

CIRCARDIAN RHYTHM[36]

There is a PM peak of SCD in ischemic cardiomyopathy, PRAISE trial also found that there is a uniformity in sudden cardiac death in non ischemic and that PM peak of ischemic cardiomyopathy mortality is not altered by antiischemic and antithrombotic therapy.

DIASTOLIC DYSFUNCTION

The prognosis less defined in studies, some shows mixed results

EFFECT OF TREATMENT

SOLVD trial of NYHA classII and III, showed decrease in four year mortality rate with the use of enalapril from 42 to 36%

In MERIT-HF trial use of ACE inhibitor , digoxin in NYHA class II and III patients addition of metoprolol have a significant reduction in one year mortality.

PROCESS OF CARE MEASURES

Improved outcomes in outpatient process-of-care measures, as observed by an observational study of 15,177 patients with left ventricular ejection fraction ≤35 percent and chronic heart failure or post-myocardial infarction. Two-year survival was improved with process measures including adherence to evidence-based drug and device therapies.

CAUSE OF DEATH IN HEART FAILURE[37,38,39]

1. Sudden cardiac death
2. Progressive heart failure

Sudden death, progressive pump failure, sudden death during worsening account for approximately one-third of death.

Ventricular tachycardia converting to ventricular fibrillation common cause of death.

Angiotensin converting enzyme, beta blocker, aldosterone antagonists improve the survival. ICD arm showed promising decreasing mortality.

ROLE OF ISCHEMIA[40]

It is underestimated in sudden cardiac death.

NYHA functional class

NYHA Grading	Functional Capacity
Class I	No limitations. Ordinary physical activity does not cause undue fatigue, dyspnoea or palpitations (asymptomatic LV dysfunction)
Class II	Slight limitation of physical activity. Ordinary physical activity results in fatigue, palpitation, dyspnoea or angina pectoris (mild CHF).
Class III	Marked limitation of physical activity. Less than ordinary physical activity leads to symptoms (moderate CHF).
Class IV	Unable to carry on any physical activity without discomfort. Symptoms of CHF present at rest (severe CHF).

With 6 and 12 **month** mortality rates of 44 and 64 % in patients with NYHA class IV.

Left ventricular ejection fraction

Symptoms of systolic heart failure starts when ejection fraction declines below 35 to 40%. Symptoms does not depend on ejection fraction. Very low ejection fraction like 20% may be asymptomatic

As an univariate predictorof prognosis is not useful, if used with NYHA class it is best predictor of prognosis.

Concomitant diastolic dysfunction

Mitral flow velocity is used to detect diastolic dysfunction Difference in changes during nitroprusside infusion like

- Irreversible restrictive, unstable restrictive, reversible restrictive, stable restrictive patterns are observed.
- Each having 51%, 33%,19%, 6% cardiac event rate
- Effect of dobutamine is observed
- Restrictive pattern causes elevation of left atrial pressure and decrease in response to ionotropins

Right ventricular function

Independent risk in survival of patients in heart failure is Right ventricular systolic dysfunction

Pulmonary artery pressure

Each 5 mmHg raise in mean pulmonary artery pressure from base line, the mortality with and without myocarditis is increased.

Other echocardiographic findings

Others include

- LV end-diastolic volume index >120 mL/m2 /LV dilatation
- LV systolic diameter index >2.75 cm/m2 and restrictive mitral filling pattern
- Increased LV mass
- Increased left atrial size

QRS prolongation and LBBB

1. qrs duration >120ms
2. Left bundle branch predicts poor outcome

Exercise variables

Peak VO2, six-minute walk distance, and exercise hemodynamics have been correlated with patient survival.

Signs of reduced tissue perfusion

A low mean arterial blood pressure, renal insufficiency, an attenuated response to diuretics, and lack of hemodynamic improvement with therapy, as indicated by failure to reduce LV filling pressure, are also associated with a poorer prognosis

Reduced myocardial blood flow

In patients with idiopathic cardiomyopathy, reduced myocardial blood flow, as assessed by positron emission tomography at rest and after intravenous dipyridamole , is an independent predictor of subsequent cardiac event.

Atrial fibrillation

Prevalence is high and dilemma weather it is an independent factor or associated with worsening of heart failure

Physical findings

S3 gallop and jugular venous pressure

Its presence has a prognostic significance.

Functional MR

Functional mitral regurgitation may be moderate to severe and is associated with poor prognosis

C-reactive protein is a survival guide in ischemic cardiomyopathy

Heart rate variability

Reduced heart rate variability indicates increased sympathetic or reduced parasympathetic tone has poor prognosis

Planar QRS-T angle

Nonsustained VT or frequent ventricular ectopy, a planar QRS-T angle more than 90 degrees was a predictor of the composite end point of total mortality

Troponins

High troponin T (TnT) and troponin I (TnI) have a poor prognosis

Low serum cholesterol

Possibly due to statin therapy

Hyperuricemia

May be due to diuretic therapy or due to low cardiac output state

Studies shown that allopurinol improves endothelial function and blood flow.

It is also proved as a risk factor for coronary artery disease

Weight loss and BMI

Patients with BMI <30 kg/m2 higher mortality than with BMI within 30 and 34.9 kg/m2

High plasma adiponectin indicates wasting so correlated with poor prognosis.

Although obesity associated with coronary heart disease but it does not correlate with mortality in heart failure

Hypoalbuminemia and liver function abnormalities

Hypoabubimemia depends on

nutritional status,

hepatic function,

hemodilution,

inflammatory state

elevated alkaline phosphatase, elevated alanine aminotransferase elevated aspartate aminotransferase are seen in an multivariate analysis total bilirubin is a better tool for prognosis in heart failure

Anabolic hormones

Decreased anabolic steroid and isulin like growth factor is associated with poor prognosis in men

Albuminuria

It has poor prognostic marker it is associated more often with class III and IV heart failure

Depression

Depression appears to be both relatively common and associated with a worse prognosis in patients with heart failure

mIBG imaging

Metaiodobenzylguanidine (mIBG) is an analog of norepinephrine (NE) that is taken up into cardiac sympathetic nerve endings in the same manner as NE. This uptake is decreased in HF. Radionuclide scintigraphy using I-123-mIBG correlates with established indices of HF severity, including LVEF, CI, PCWP and peak VO2

COMORBIDITIES

ANEMIA[41]

Etiology:

1. Increased cytokines in circulation
2. Dilutional anemia[42]
3. Iron deficiency[43,44]
4. Use of angiotensin converting enzyme inhibitor[45]
5. Cardiorenal syndrome[46,47,48]

Development of high output heart failure

Hemoglobin less than 5g/L produces failure in the absence of underlying heart failure

One study showed , each 1gm/dL decrease has independent association with left ventricular dilation.

IMPACT OF ANEMIA ON MORTALITY[50]

1. Worse hemodynamics[49], high urea nitrogen in blood, low albumin
2. Greater NYHA classIV occurrence
3. Low peak oxygen consumption

It is an independent risk factor for mortality in heart failure patients

IMPACT OF POLYCYTHEMIA[51]

It is also increase the mortality, correlate to presence of COPD

TREATMENT OF ANEMIA[52,53]

Intravenous iron can be used and provide symptomatic relief in hemoglobin below 13 gm/dl and low ferritin(<100microgram/L) and transferrin saturation(<20%)

DIABETES AND HF[54]

Framingham study showed a epidemiological link between diabetes and heart failure

Factors associated are[55-57,58,59]

1. Age
2. Duration of DM
3. Insulin use
4. Ischemic heart disease
5. PVD(peripheral vascular disease)
6. Raised serum creatinine
7. Poor glycemic control
8. Microalbuminuria
9. increase in baseline fasting plasma glucose associated with increase risk of HF

10. thiozolindinediones and metformin have toxicities that worsens HF
11. diabtetes perse causes diastolic and systolic dysfunction leads to diabeteic cardiomyopathy
12. AHA guideline suggests avoidance of thiozolindinediones and metformin inNYHA class III and IV dyspnoea[60-66]

PREDICTORSOF HOSPITAL READMISSION WITH HEART FAILURE

Heart failure has emerging as the leading cause of hospitalization. Ischemic etiology is known cause of mortality and readmission in heart failure patients. Powerful predictor of prognosis is left ventricular function,

Krumoltz et all examined readmission in patients with heart failure, he found four major predictors

1. Prior history of hospitalization within one year
2. Prior heart failure history
3. Elevated renal parameters >2.5 during prior discharge
4. Co-morbid illness like diabetes mellitus

Other studies showed the predictors of outcome during admission are

1. Hemodynamic abnormalities
2. Plasma atrial natriuretic peptide

3. Inotropic reserve
4. Plasma epinephrine
5. Total oxygen consumption during peak exercise

LACE INDEX SCORE OF READMISSIONS

SCORE of 10 or more is used to segregate the patients with high risks

Attribute	Value	Points
Length of Stay, d (**L**)	<1	0
	1	1
	2	2
	3	3
	4-6	4
	7-13	5
	≥ 14	7
Acute admission (**A**)	Yes	3
Comorbidity (Charlson index) (**C**)	0	0
	1	1
	2	2
	3	3
	≥ 4	5
Visits to ER in past 6 months (**E**)	0	0
	1	1
	2	2
	3	3
	≥ 4	4

MATERIALS AND METHODS

MATERIALS AND METHODS

SELECTION OF PATIENTS:

Patients admitted for heart failure previously in madras medical college and Rajiv Gandhi government general hospital and now readmitted with history of heart failure symptoms are included in the study. On admission 10cc blood is withdrawn from the patient after obtaining the informed consent either from the patient or the relatives. The sample is tested for complete blood count, renal function tests, liver function tests and serum electrolytes. As this study is both prospective and retrospective the lab parameters and clinical parameters of patients previously admitted are obtained from medical records department, Rajiv Gandhi government general hospital.

STUDY CENTRE:

Institute of Internal Medicine,

Madras Medical College and Rajiv Gandhi Government General Hospital, Park town, Chennai-600003.

DURATION OF THE STUDY:

6 months(April 2014 – September 2014)

STUDY DESIGN:

Prospective and Retrospective observational study

SAMPLE SIZE:

98 PATIENTS

DATA COLLECTION AND METHODS:

Patients are subjected to history questioning, clinical examinations and blood sampling. Retrospective samples are obtained from case sheet records at medical records department.

PROCEDURE / INVESTIGATION DETAILS:

1. Complete Blood Count
2. Renal Function Test – Urea, Creatinine
3. Serum Sodium
4. Serum Potassium
5. ECG
6. Echocardiogram
7. Chest X Ray
8. Pulse Rate
9. Blood Pressure.

INCLUSION CRITERIA :

1. Age: above 15 years.
2. Sex-both genders.
3. Patients presenting with symptoms of heart failure according to Framingham criteria.
4. Patients willing to give written informed consent.

EXCLUSION CRITERIA :

1. Patient age less than 15 years
2. Patient who was previously admitted for symptoms of heart failure and now admitted for another cause

SPONSORSHIP :

NO

CONFLICT OF INTEREST :

None

OBSERVATION

OBSERVATION AND RESULTS

Table 1: Showing age wise distribution of study population

Age	Patients	Percentage
15-29	4	4%
30-44	19	19.3%
45-55	44	44.8%
>55	31	31.6%

Figure 1: showing age wise distribution of study population

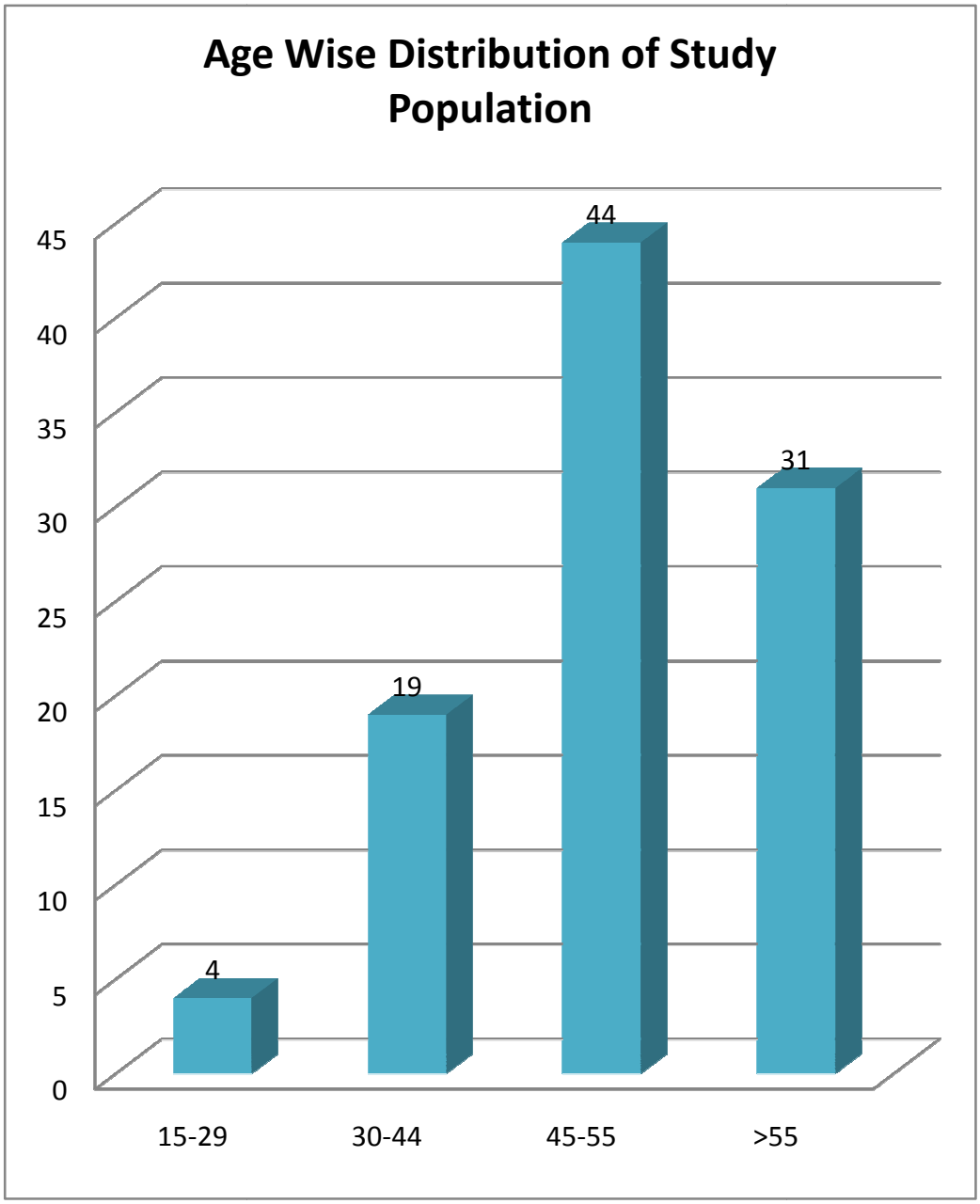


Table 2: showing age wise distribution of study population

Sex	No of Patients	Percentage
Male	58	59.1%
Female	40	40.9%

Figure 2: Showing sex wise distribution of study population

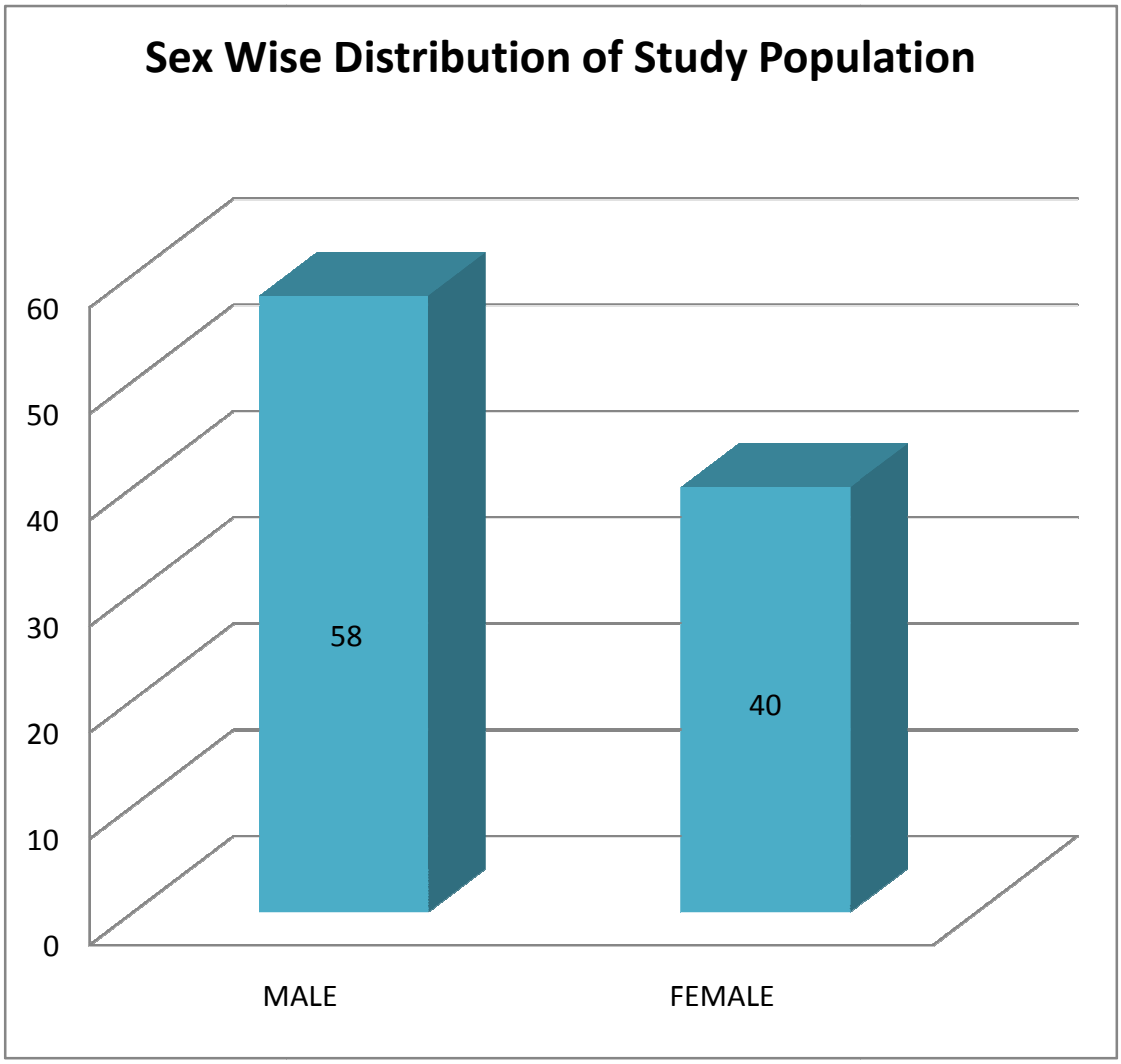


Table 3: Showing bilirubin elevation in study population

Total Billirubin	No of Patients	Percentage
Elevated	6	6.1%
Not Elevated	92	93.9%

Figure 3: Showing bilirubin elevation in study population

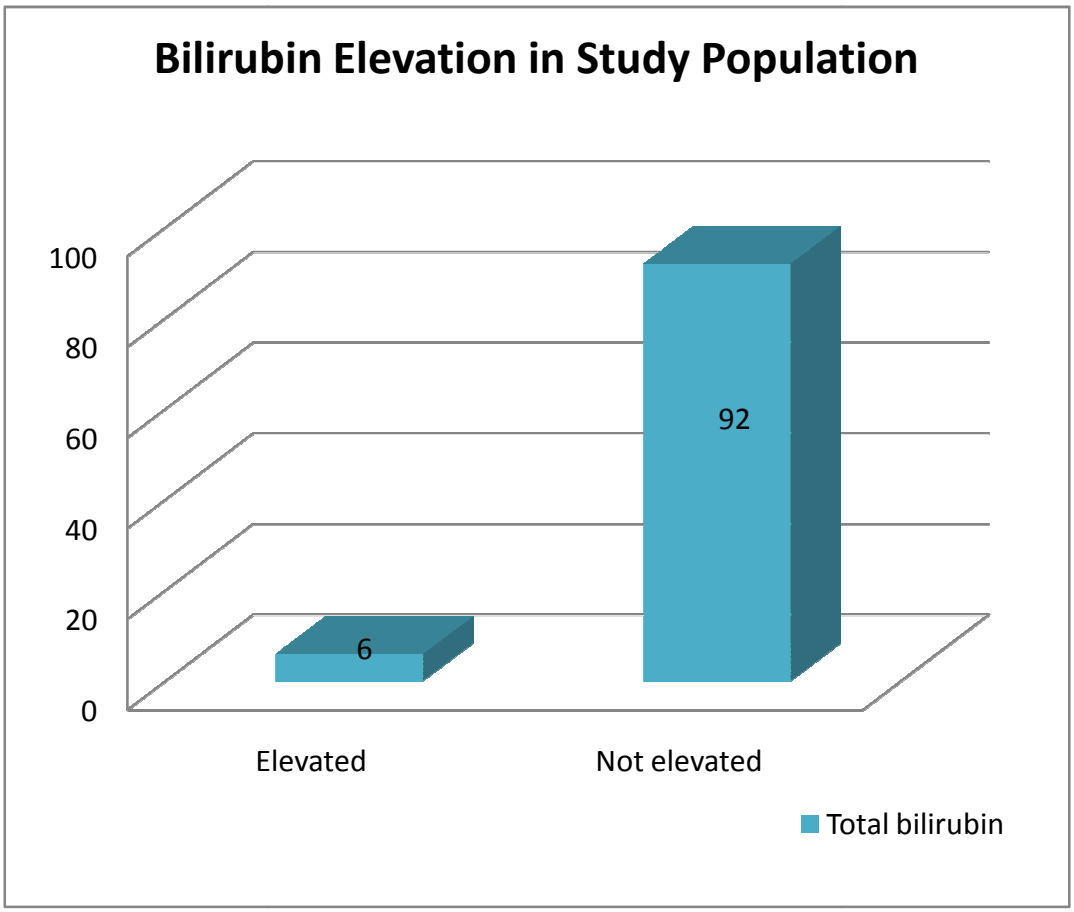


Table 4: Showing duration of stay in the study population

Days	No. of Patients	Percentage
1-4 DAYS	31	31.63%
5-9 DAYS	45	45.91%
>10 DAYS	22	22.40%

Figure 4: showing duration of stay in the study population

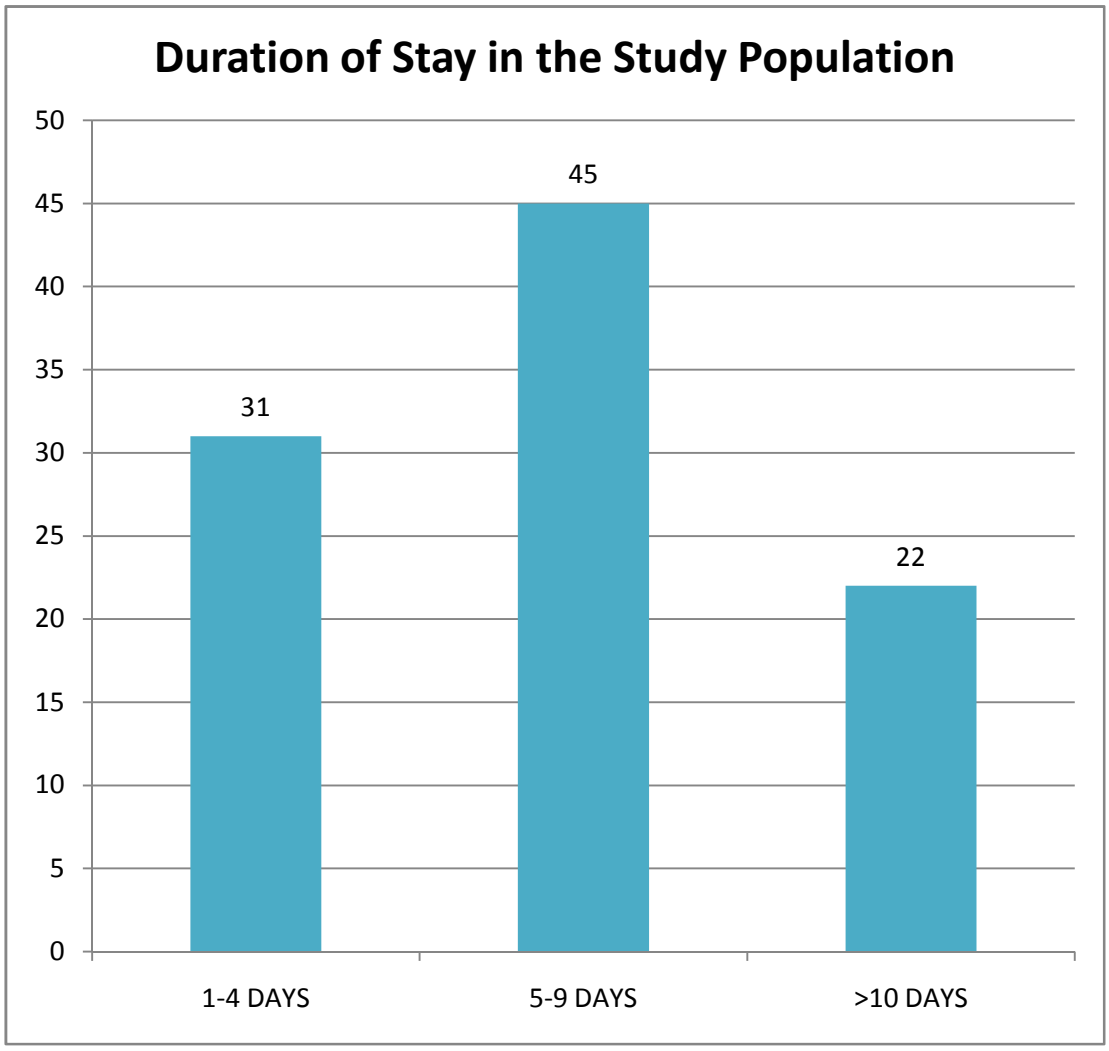


Table 5 : Showing Underlying Cause for heart failure in study population

Underlying Cause	No of patients	Percentage
1	8	8%
2	35	35.70%
3	11	11.20%
4	11	11.20%
1,2	17	17.30%
1,3	1	1%
1,4	1	1%
2,4	3	3%
4,5	11	11.20%

UDERLYING CAUSES

Hypertensive heart disease-1

Ischemic heart disease-2

Other cardiomyopathies-3

Valvular heart disease-4

Atrial fibrillation or flutter-5

Figure 5 : Showing Underlying Cause for heart failure in study population

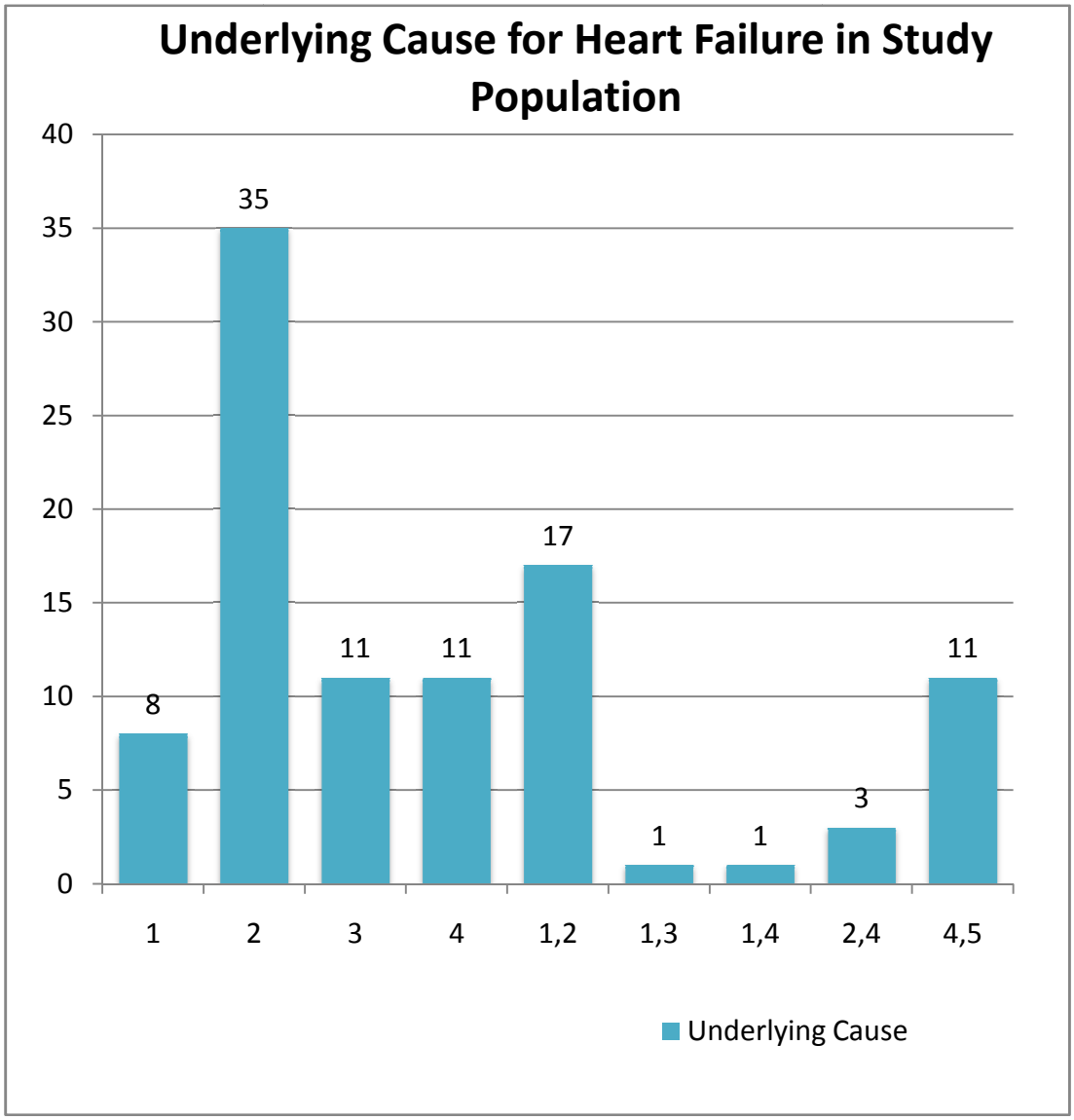


Table 6 : Showing comorbid illness in study population

Comorbid illness	No of patients	Percentage
1	7	7.10%
2	3	3%
3	26	26.50%
4	13	13.20%
5	6	6.10%
1,2	1	1%
1,3	3	3%
1,4,5	1	1%
1,5	1	1%
3,4	1	1%

1- Acute or chronic renal disease

2-Chronic lung disease

3-Diabetes mellitus

4-Anemia

5-Drug or alcohol abuse

Figure 6 : Showing comorbid illness in study population

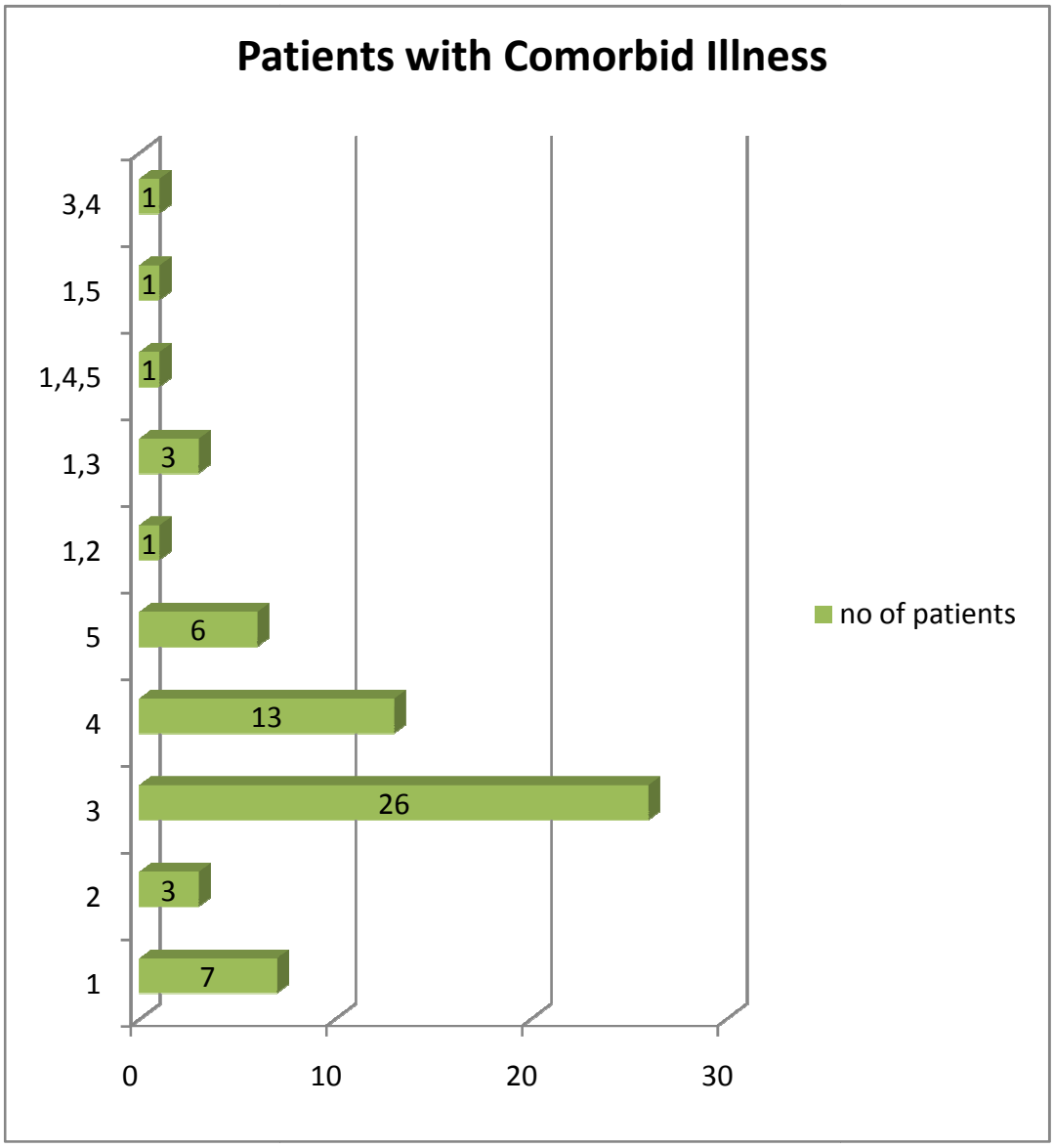


Table 7 : Showing precipitating Factor in study population

Precipitating Factor	No of patients	percentage
1	15	15.30%
3	5	5.10%
4	2	2%
5	1	1%
6	2	2%
7	12	12.20%
8	5	5.10%
1,2	2	2%
1,6	1	1%
1,7	5	5.10%
1,7,8	1	1%
1,8	2	2%
3,6	1	1%
4,7	1	1%
5,7	2	2%

1-lack of compliance

2-uncontrolled hypertention

3-cardiac arrhythmias

4-Inadequate therapy

5-pulmonary infection

6-emotional stress

7-inappropriate medication or fluid overload

8-myocardial infarction

9-endocrine disorders

Figure 7 : Showing precipitating Factor in study population

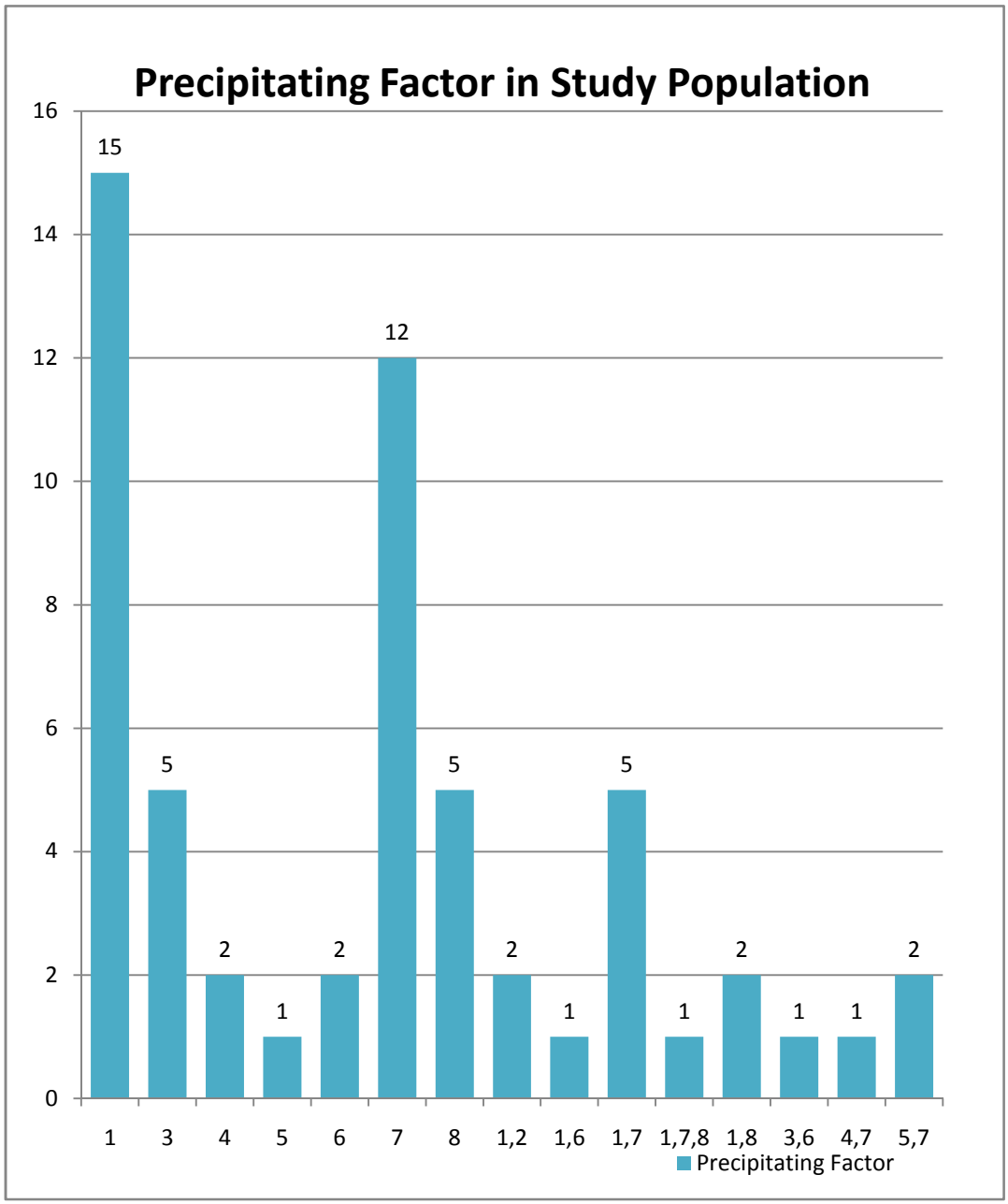


Table 8 : Showing CXR findings in study population

CHEST X RAY	No of Patients	Percentage
Pulmonary hypertension grade I according to larry Elliot's classification upper lobe veins prominent	36	36.70%
Grade II PHT either kerley A,B,C lines or hilar haziness	14	14.20%
GradeIII PHT- bilateral patchy opacities	12	12.20%
Pleural effusion	2	2%
Postcapillary pulmonary arterial hypertension showing main pulmonary artery dilation	22	22.40%
Precapillary pulmonary arterial hypertension	2	2%
Normal lung field	9	9.10%

Figure 8 : Showing CXR findings in study population

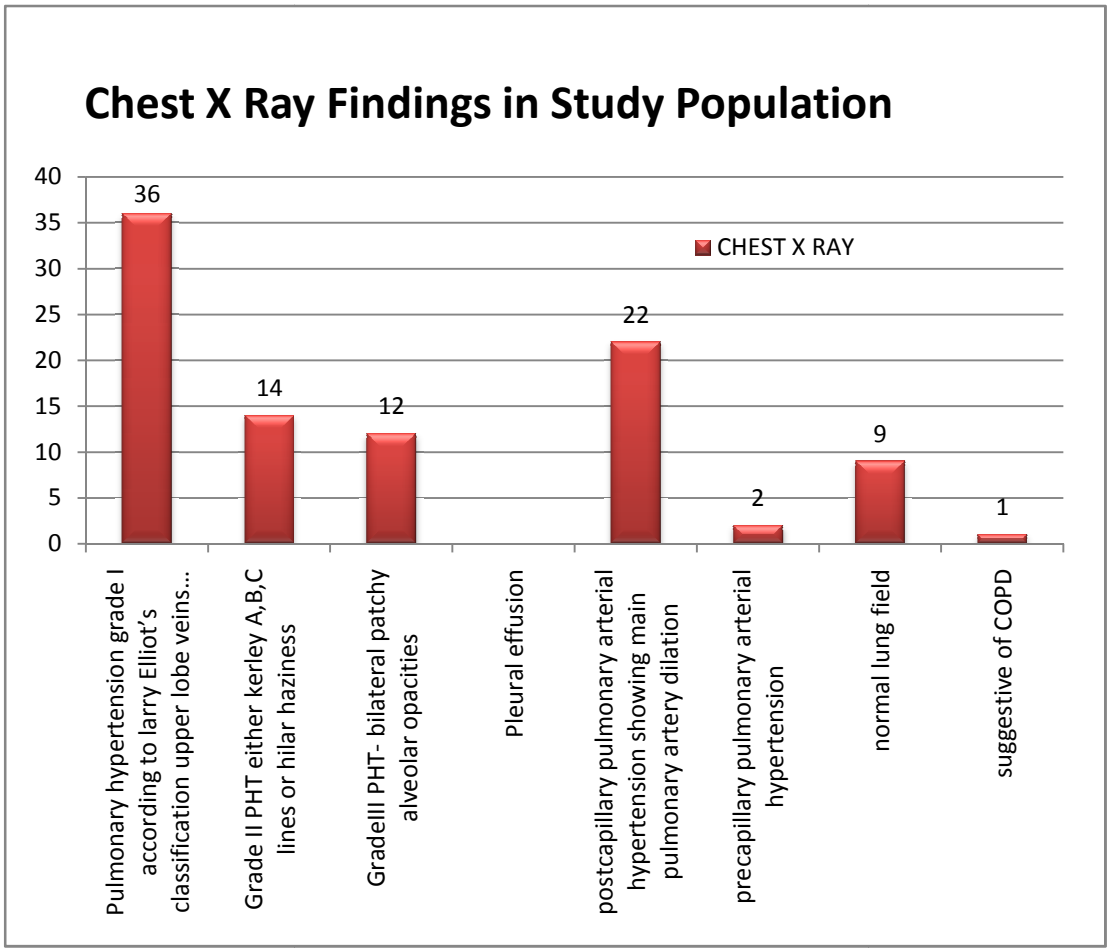
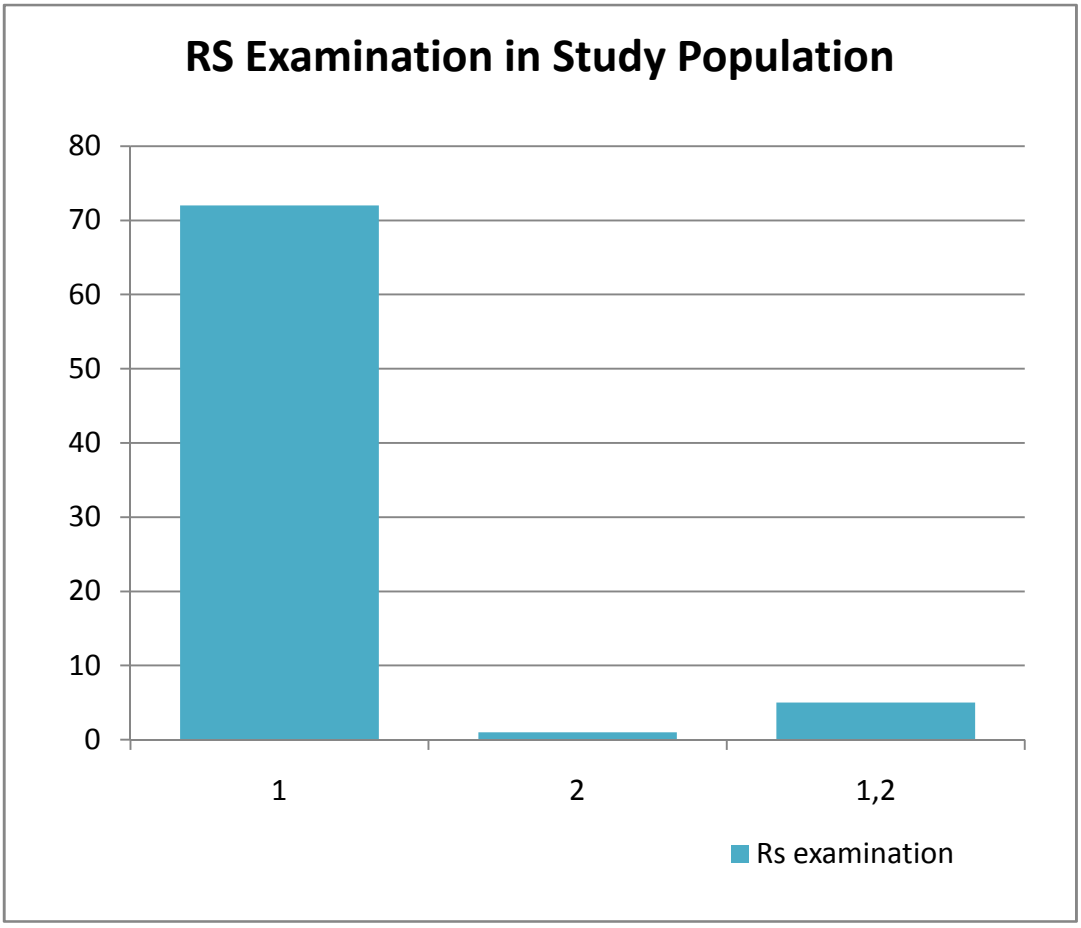


Table 9 : Showing RS examination in study population

RS examination	No of patients	Percentage
1	72	73.40%
2	1	1%
1,2	5	5.10%

Figure 9 : Showing RS examination in study population



1. Basal crepitations
2. Wheeze

Table 10 : Showing CVS findings in study population

CVS findings	No of patients	Percentage
S3 heard	28	28.50%
Systolic murmur heard	8	8.10%

Figure 10 : Showing CVS findings in study population

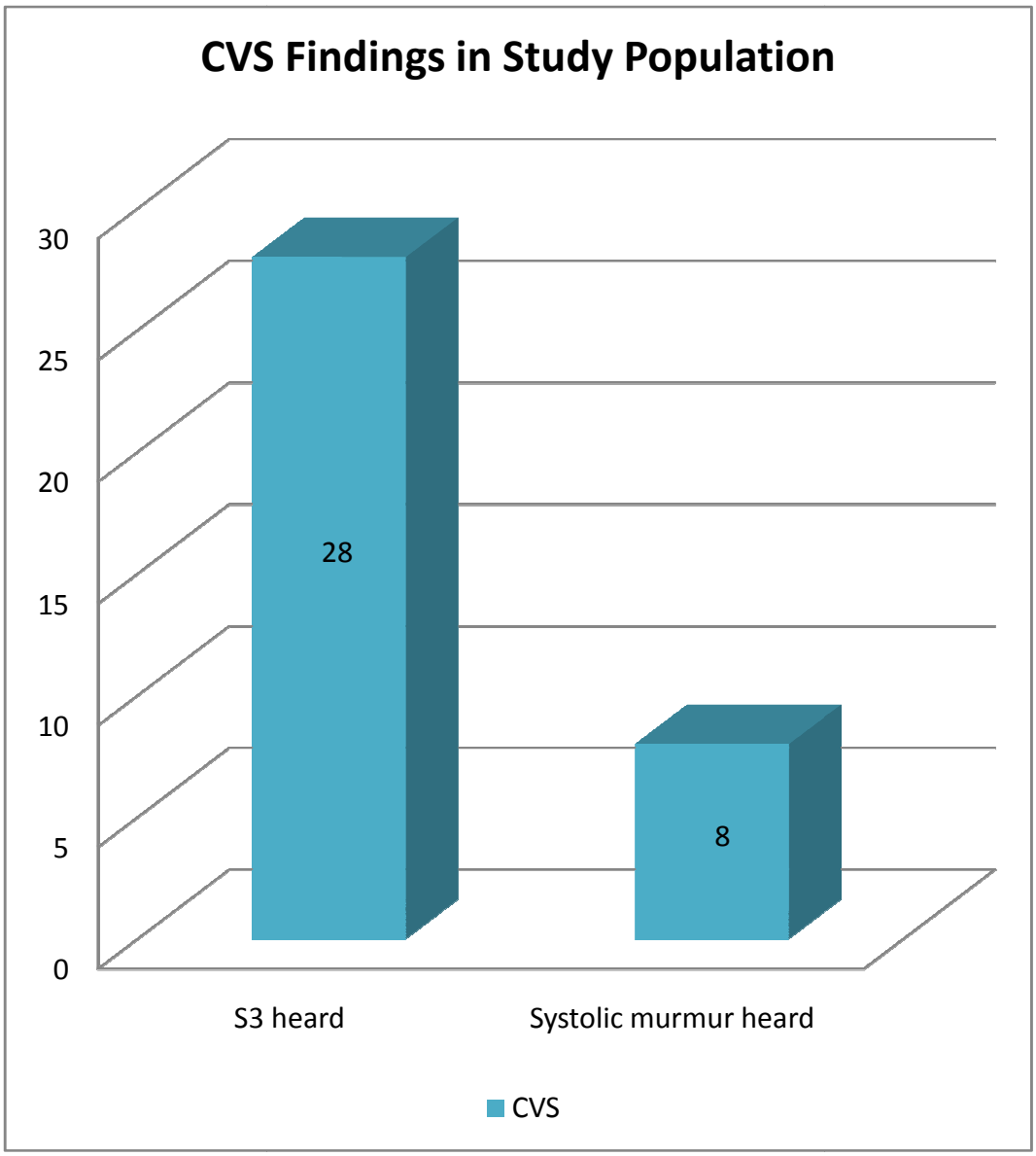


Table 11 : Showing echo findings in study population

ECHO findings	No of Patients	Percentage
3	2	2%
4	12	12.20%
5	8	8.10%
6	6	6.10%
7	7	7.10%
8	8	8.10%
1,3	10	10.20%
1,4	3	3%
1,6	1	1%
1,7,9	1	1%
1,8	1	1%
2,3	1	1%
3,7	1	1%
4,5	1	1%
4,8	4	4%
5,10	1	1%
5,4	1	1%
5,8	4	1%
5,9	1	1%
6,9	2	1%
7,1	2	1%
7,10	1	1%
7,4	2	2%
7,8	2	2%
7,9	15	15.30%

1. Valvular diseases
2. Interatrial and interventricular shunts
3. Right ventricular dilation and D shaped interventricular septum
4. Left ventricular diastolic dysfunction
5. Left ventricular systolic dysfunction EF; 41- 45%
6. Left ventricular systolic dysfunction EF; 36-40%
7. Left ventricular systolic dysfunction EF; 30-35%
8. Regional wall motion abnormality
9. Global hypokinesia
10. Pericardial disease

Figure 11 : Showing echo findings in study population

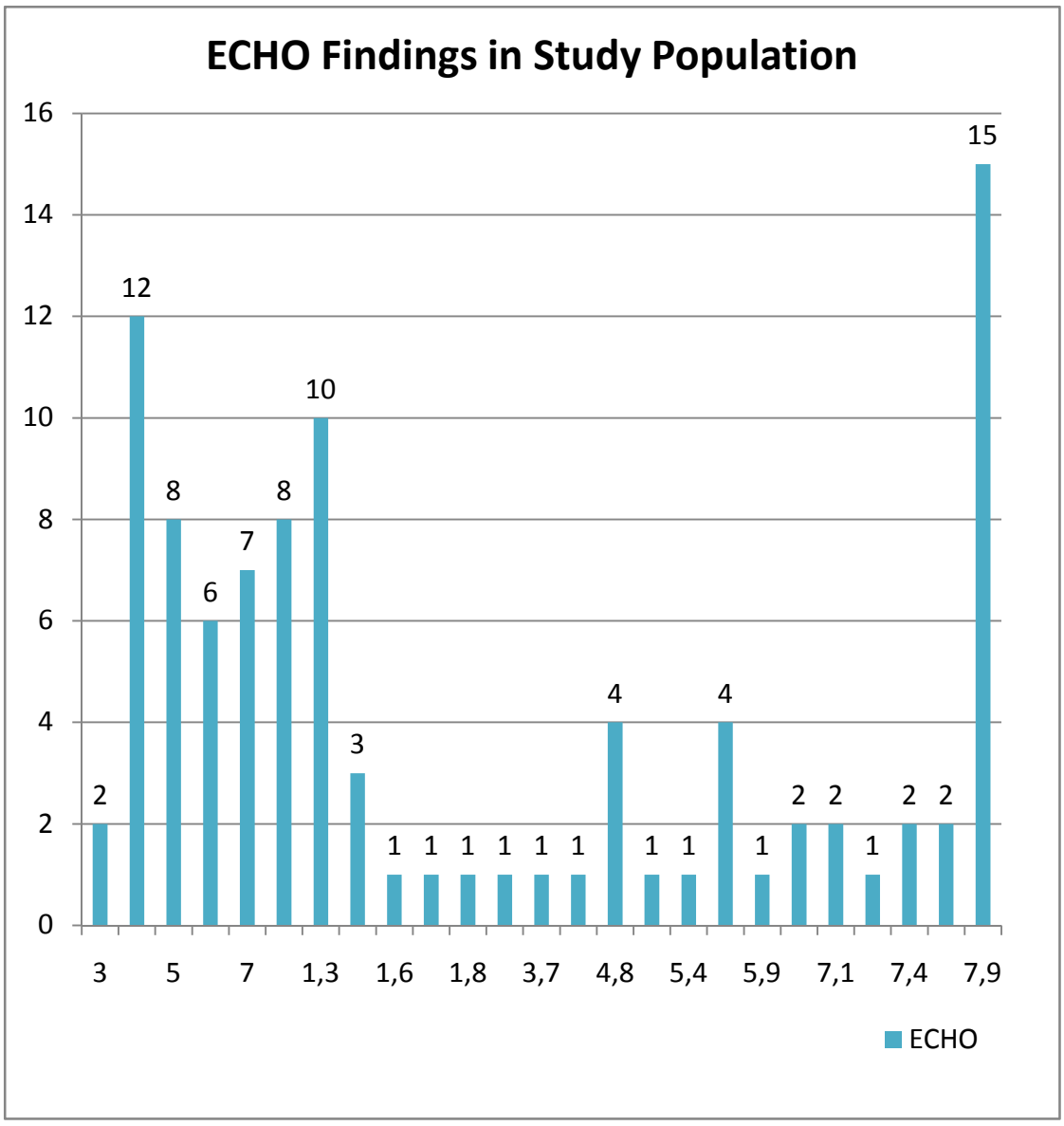


Figure 12 : Showing distribution of hemoglobin in study population

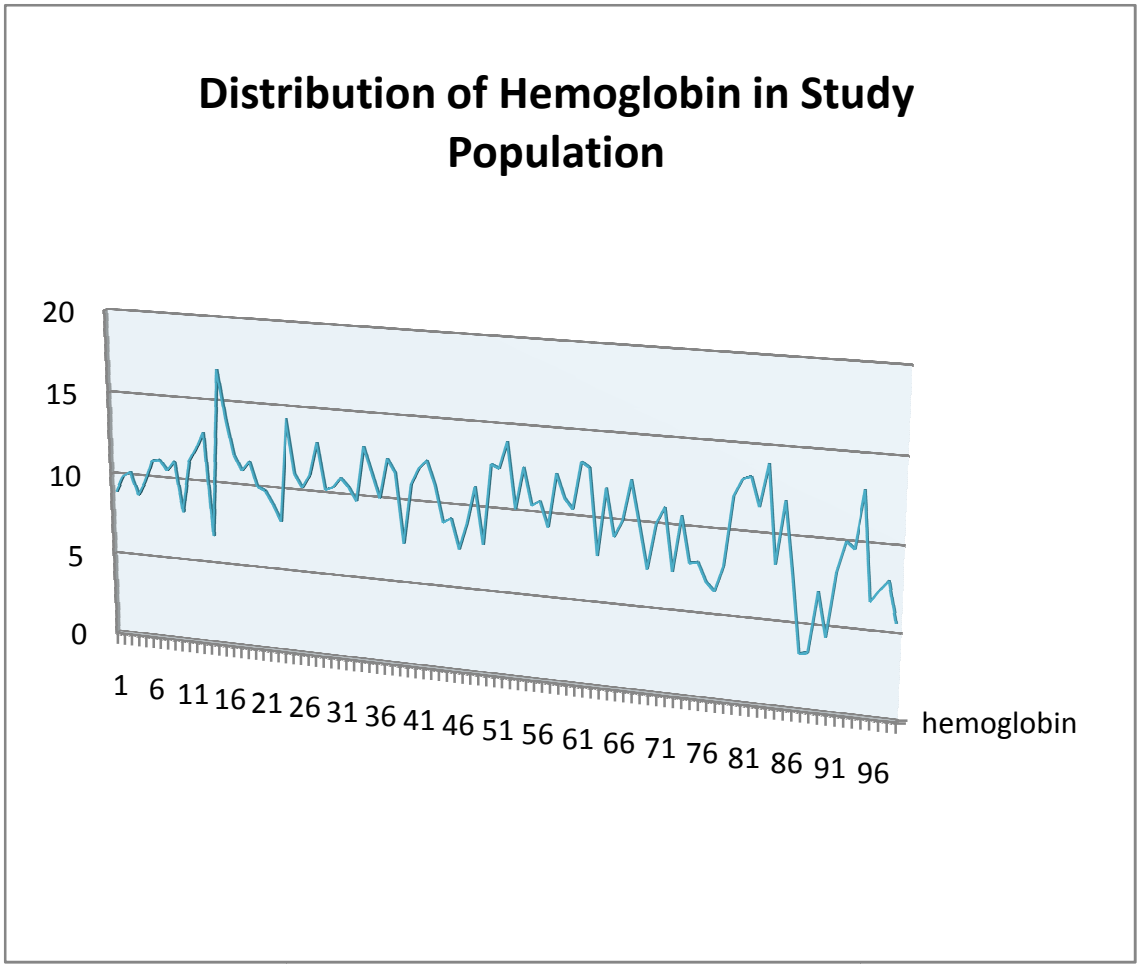


Figure 13 : Showing distribution of creatinine in study population

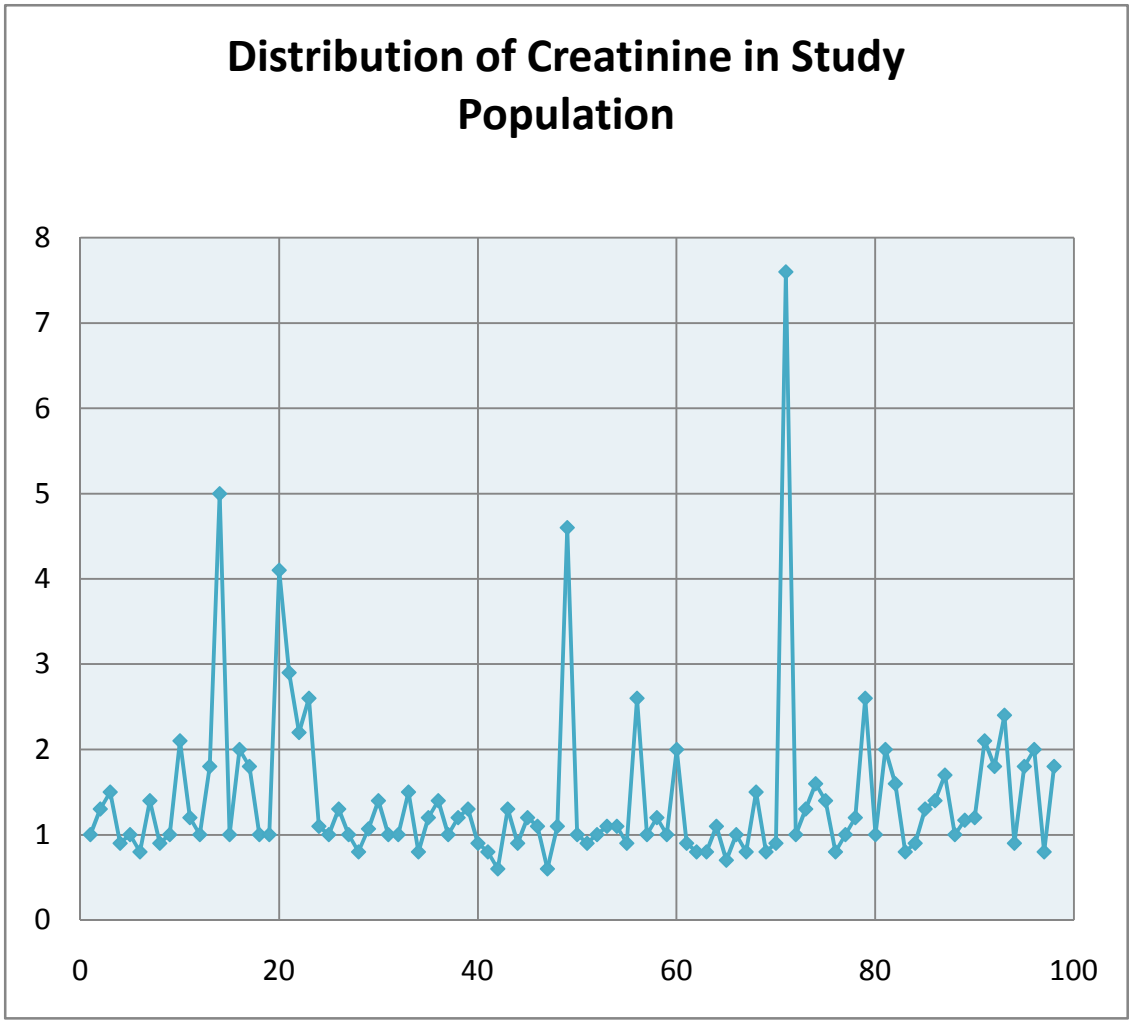


Figure 14 : Showing distribution of potassium in study population

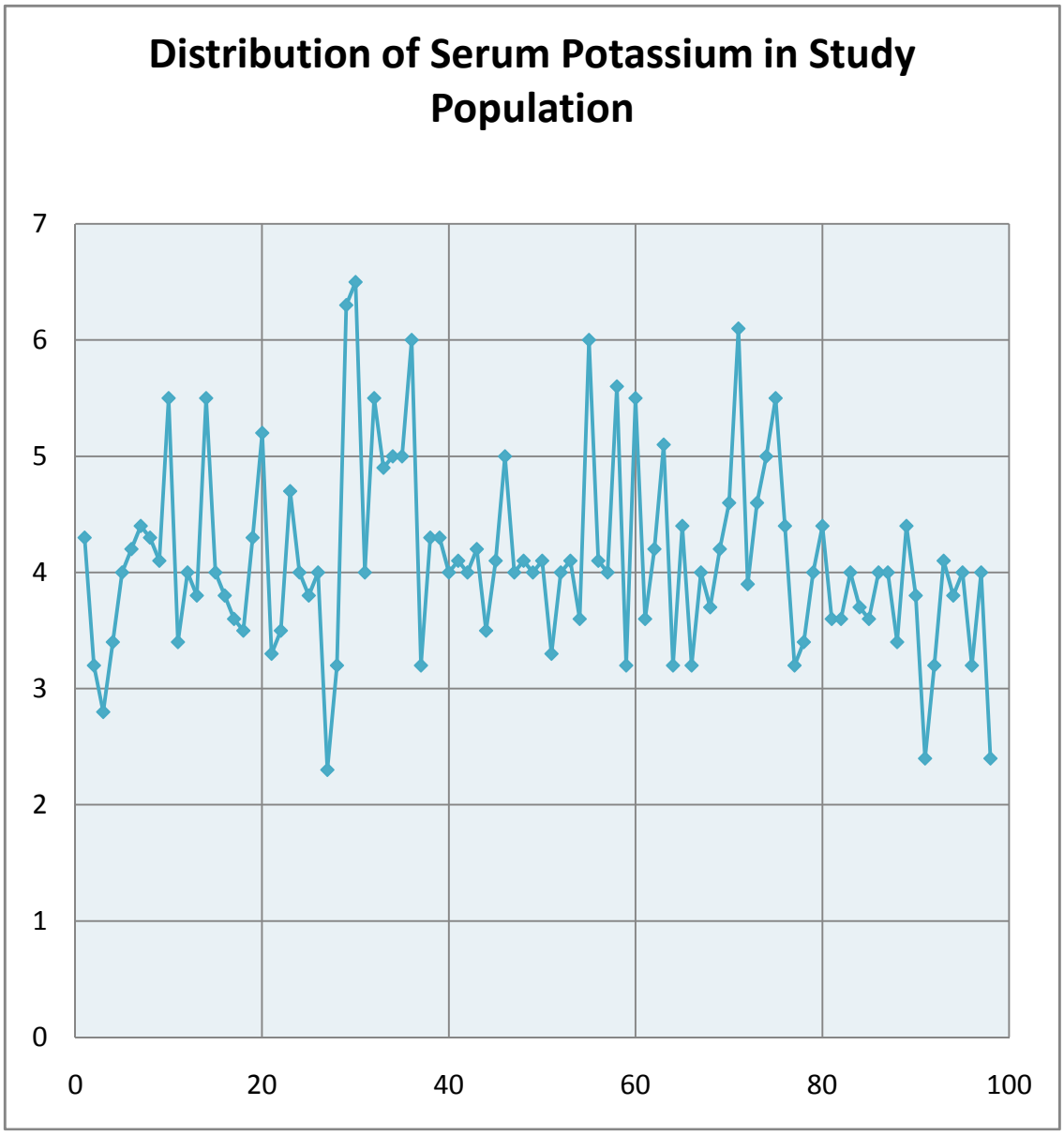


Figure 14 : Showing distribution of urea in study population

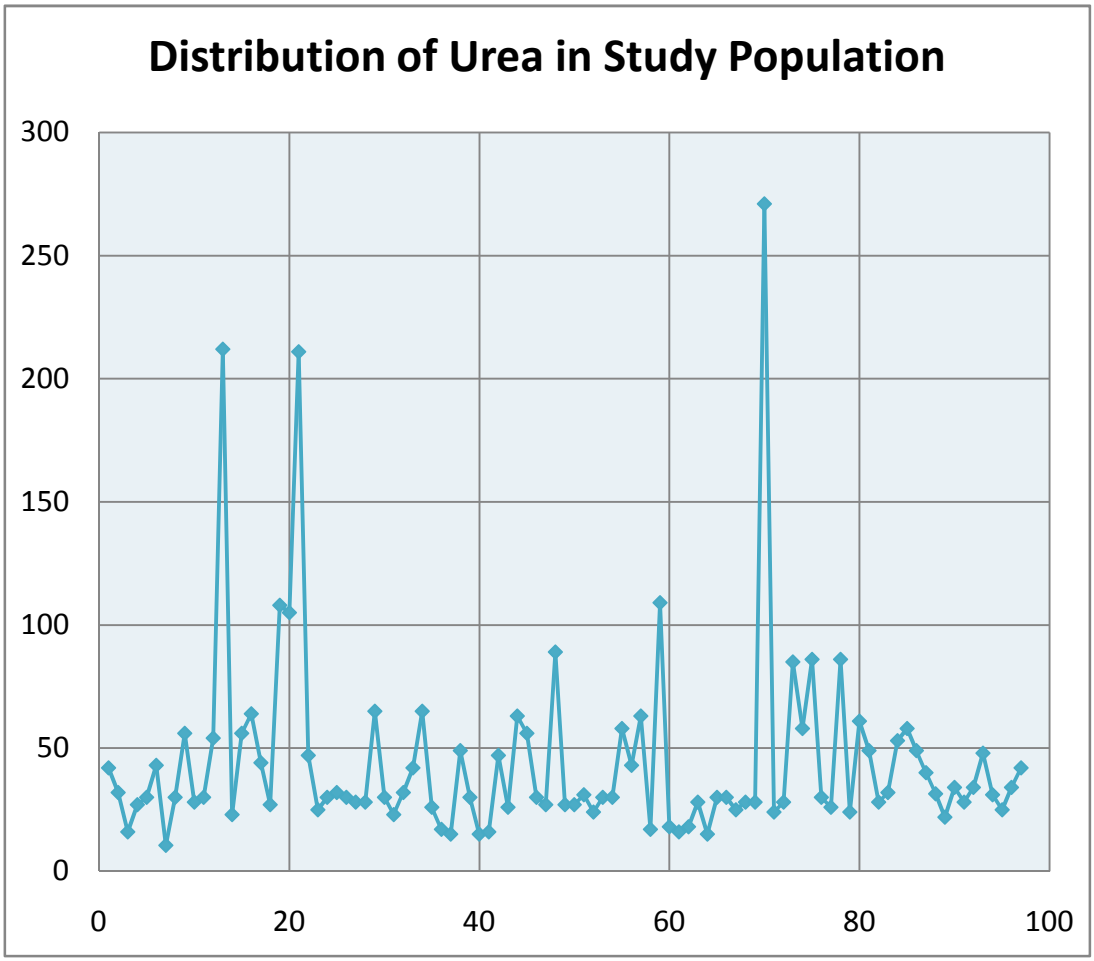


Figure 15 : Showing ECG findings in study population

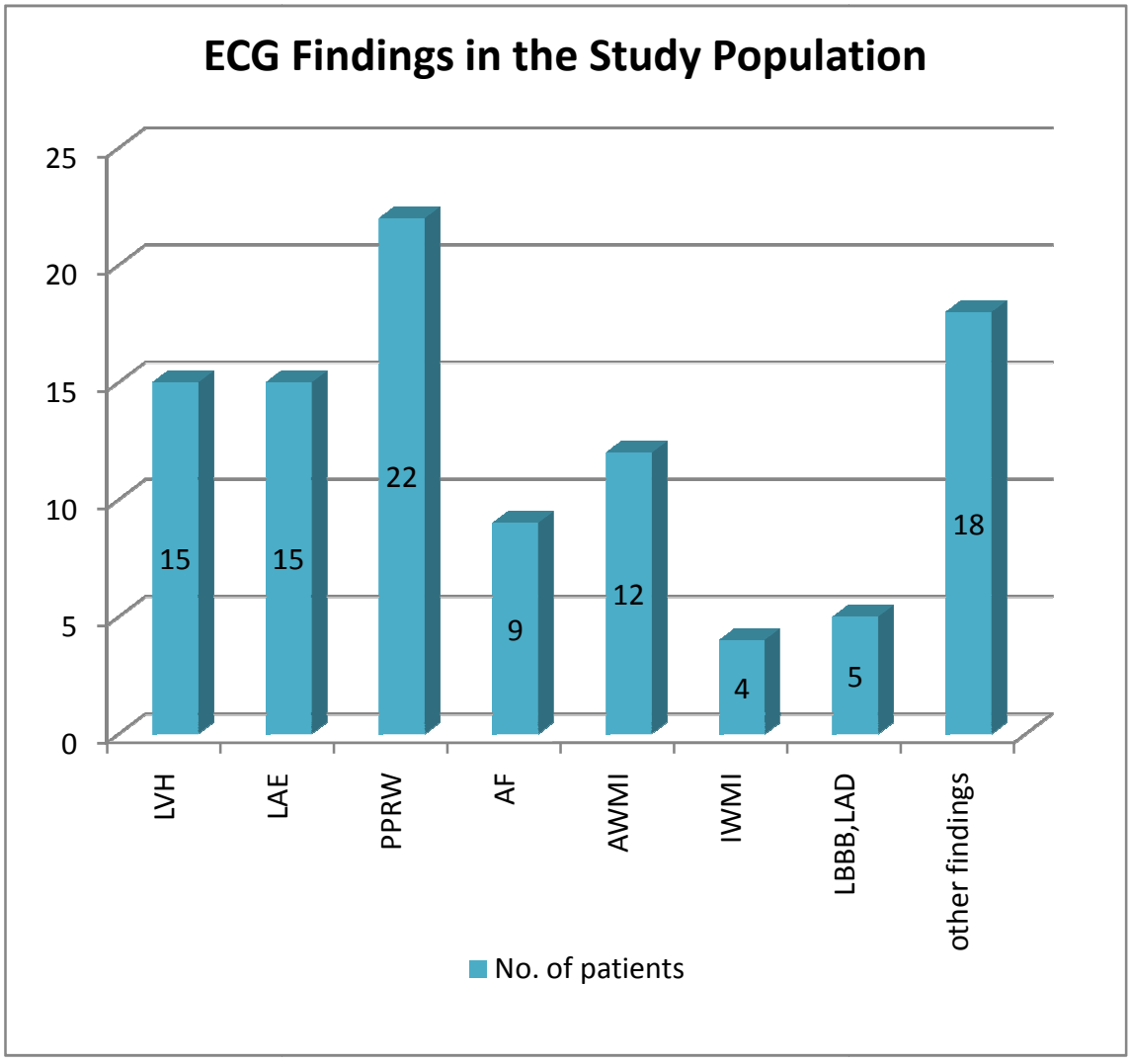


Table 12 : Showing ECG findings in study population

ECG finding	No. of patients	Percentage
LVH	15	15.20%
LAE	15	15.20%
PPRW	22	22.30%
AF	9	9%
AWMI	12	12.20%
IWMI	4	4%
LBBB,LAD	5	5%
other findings	18	18.30%

LVH- left ventricular hypertrophy

LAE- left atrial enlargement

PPRW- poor progression of R wave

AF- atrial fibrillation

AWMI- anterior wall myocardial infarction

IWMI- inferior wall myocardial infarction

LBBB, LAD- left bundle branch block, left axis deviation

DISCUSSION

DISCUSSION

This study is a prospective and retrospective observational study and was done in Madras Medical college and Rajiv Gandhi government general hospital. During the study 100 patients are selected and two patients did not give consent for the study, then 98 patients with the diagnosis of cardiac failure who were admitted earlier in this hospital and readmitted within 3 months of discharge were included in the study. Informed consent was obtained for the investigations that were required during the present admission.

The acute heart failure syndrome is one of the major burden of hospital admissions. The problem of readmission in cardiac failure patients is increasing. About more than half of the patients had coronary artery disease and almost half of the patient had valvular heart disease, which were correctable causes. Due to lack of infrastructure in a developing country like india it makes the treatment impossible even for those in whom it is treatable. Programmes that alter the patient behavior towards the disease and modify the risk factors and risk behaviors like alcohol intake should be implemented.

AGE AND HEART FAILURE

Our study showed that the heart failure is occurring in relatively younger age group than in other studies conducted in various hospitals. The Framingham study showed that the incidence and prevalence of heart failure increases steeply with age, but in MMC RGGGH readmissions were common in the age group of 40 to 54 years .

GENDER AND HEART FAILURE

Vaccarino et al showed women can tolerate heart failure better than men because the left ventricular systolic function and high mean arterial pressure is better preserved in these patients. Makene et all study showed male to female ratio of 1:2. Our study showed 60% of males and 40% females in medical wards. In peripartum cardiomyopathy, 59% of patients recover spontaneously, 26% of patients show recurrence in subsequent pregnancies, 9% experience progressive dilated cardiomyopathy and 11% die within the first year. In our study only 3 patients had peripartum cardiomyopathy.

AETIOLOGY OF HEART FAILURE

As heart failure can involve both the ventricles, it mostly affects the left ventricle. Isolated right heart failure does not occur except in cor pulmonale.

Etiology has the following 3 components

1. Underlying cause
2. Comorbid illness
3. Precipitating factor

Most common cause in most of the studies is coronary artery disease (CAD). In our study CAD tops the list and then follows the valvular heart disease. Most common comorbid illness is diabetes mellitus which is then followed by anemia and renal failure. Most common precipitating factor is lack of compliance towards the drugs and then follows lack of fluid and salt restriction. Comorbid illness do not directly attribute to failure, they do by precipitating factor to set in.

In a study of home monitoring system dyspnoea and light headedness are considered as the common symptoms that has highest frequency of readmissions. Anemia is invariably present in all patients.

In echocardiogram systolic dysfunction is the most common cause for readmission, then follows the right ventricular dysfunction due to valvular heart disease and the diastolic dysfunction.

DURATION OF STAY

Length of stay in 46% of patients who were admitted during previous admissions is within 5 to 10 days. 22.4% percent of patients had more than 10 days of stay and 31% of patients had less than 5 days of stay. Two patients died in the next admission in whom there was longer the length of stay. But the patients who were discharged within 10 days experience more frequency of readmission which may be due to the premature discharge.

SYMPTOM AND READMISSIONS

Common presenting symptom is dyspnoea on exertion as most of our patients belong to lower socioeconomic status so they depend on their physical abilities to fulfill their daily needs. Orthopnea is the equivalent symptom presenting during admission. Patients are enquired about number of pillows used during the sleep.

RADIOGRAPHIC FINDINGS

37% of patients presented with pulmonary edema grade I by larry elliot's classification. The next common finding was pulmonary arterial hypertension (main pulmonary artery dilatation) which was observed in 22.4% of our study population . Bilateral pleural effusion was observed in 2% of patients.

CARDIOVASCULAR EXAMINATION

28.9% of our study population had S3 gallop. 8% of patients had systolic murmur due to the functional mitral regurgitation.

RESPIRATORY SYSTEM EXAMINATION

73.4% of patients of our study population had basal crepitations and few had polyphonic wheeze(cardiac asthma).

ECHOCARDIOGRAM

Patients were subjected to echocardiogram within 48 hours of admission and this variable was compared with other studies. In our study left ventricular end diastolic volume and left ventricular end systolic volume was measured and cut off value was set for each variable. These were invariably increased in most of the cardiomyopathies. Most of the patients had left ventricular systolic dysfunction which was severe, 15% had mitral stenosis either alone or associated with other valvular involvement. 12% had left ventricular diastolic dysfunction with preserved left ventricular systolic function.

POOR KNOWLEDGE

Each patients were enquired about the nature of the disease and the future outcomes by brief questionnaire during the study. The observation was that the view about disease progression and the diuretic dose adjustment according to congestive symptoms are low in about 40% of the patients.

As the Indian food preparation contains high amount of sodium, the sodium restriction for these patients were also difficult.

LIVER FUNCTION TEST

Only 6% of patients showed raise in Bilirubin. These patient had symptoms of congestive hepatitis.

CONCLUSION

CONCLUSION

The inferences drawn from the study are

1. Readmissions are common in the middle age group patients.
2. Certain diseases like rheumatic valvular heart diseases are amenable to surgical correction which can prevent the heart failure.
3. Drugs that decrease the disease progression are not used appropriately as there is lack of compliance and awareness
4. Diabetes mellitus and heart failure commonly are associated in many patients
5. Severe left ventricular systolic dysfunction and diastolic dysfunction is the most common echocardiogram finding associated with heart failure.

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ANNEXURES

ABBREVIATIONS

ACC/AHA	**–**	**American College of Cardiology and the American Heart Association**
AHFS	**–**	**Acute Heart Failure Syndrome**
AT1	**–**	**Angiotensin Receptor 1**
ECF	**–**	**Extracellular Fluid**
ECG	**–**	**Electrocardiogram**
HFpEF	**–**	**Heart Failure with Preserved Ejection Fraction**
RAS	**–**	**Renin Angiotensinogen System**
HF	**–**	**Heart Failure**
IVC	**–**	**Inferior Vena Cava**
MRI	**–**	**Magnetic Resonance Imaging**
NE	**–**	**Nor Epinephrine**
ET-1	**–**	**Endothelin1**
CT	**–**	**Computerized Tomography**
OSA	**–**	**Obstructive Sleep Apnea**
SHIFT	**–**	**Ivabridine and Outcomes in Chronic Heart Failure**
DM	**–**	**Diabetes Mellitus**
ANP	**–**	**Atial Natriuretic Peptide**
BNP	**–**	**Brain Natriuretic Peptide**
ROS	**–**	**Reactive Oxygen Species**
NYHA	**–**	**New York Heart Association**
LBBB	**–**	**Left Bundle Branch Block**

PROFORMA

Causes and Prediction of Readmission of Heart Failure Patients – Questionnaire

Name : Patient ID No:

Age/Sex :

Socioeconomic status:

Marital status:

Occupation:

Underlying cause

Hypertensive heart disease: ☐

Ischemic heart disease: ☐

Other cardiomyopathies: ☐

Valvular heart disease ☐

Atrial fibrillation or flutter: ☐

Comorbid illness

Acute or chronic renal disease: ☐

Chronic lung disease: ☐

Diabetes mellitus: ☐

Anemia: ☐

Drug or alcohol abuse ☐

Precipitating factor

lack of compliance ☐

uncontrolled hypertention ☐

cardiac arrhythmias ☐

inadequate therapy ☐

pulmonary infection ☐

emotional stress ☐

inappropriate medication or fluid overload ☐

myocardial infarction ☐

endocrine disorders ☐

Duration of stay :

General examination

Systemic examination

CVS:

RS:

P/A:

CNS:

Investigations :

ECG

Echocardiogram

Chest x ray

Thyroid function test

Complete blood count

Blood biochemistry

Serum electrolytes

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. J. Sudha Mallika,
Post Graduate, MD (General Medicine)
Institute of Internal Medicine,
Madras Medical College,
Chennai – 600003.

Dear Dr. J. Sudha Mallika,

The Institutional Ethics Committee has considered your request and approved your study titled **"CAUSES AND PREDICTION OF READMISSIONS OF HEART FAILURE PATIENTS"** No. 54072014.

The following members of Ethics Committee were present in the meeting held on 01.07.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D., Dean, MMC, Ch-3.	-- Deputy Chair Person
3.	Prof. Kalaiselvi, MD., Vice-Principal, MMC, Ch-3	-- Member Secretary
4.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
5.	Dr. G. Muralidharan, Director Incharge , Inst. of Surgery	-- Member
6.	Prof. Md Ali, MD., DM., Prof & HOD of MGE, MMC, Ch-3.	-- Member
7.	Prof. Ramadevi, Director i/c, Biochemistry, MMC,Ch-3.	-- Member
8.	Prof. Saraswathy, MD., Director, Pathology, MMC, Ch-3.	-- Member
9.	Prof. Tito, Director, i/c. Inst. of Internal Medicine, MMC	-- Member
10.	Thiru. Rameshkumar, Administrative Officer	-- Lay Person
11.	Thiru. S. Govindasamy, BABL, High Court, Chennai-1.	-- Lawyer
12.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

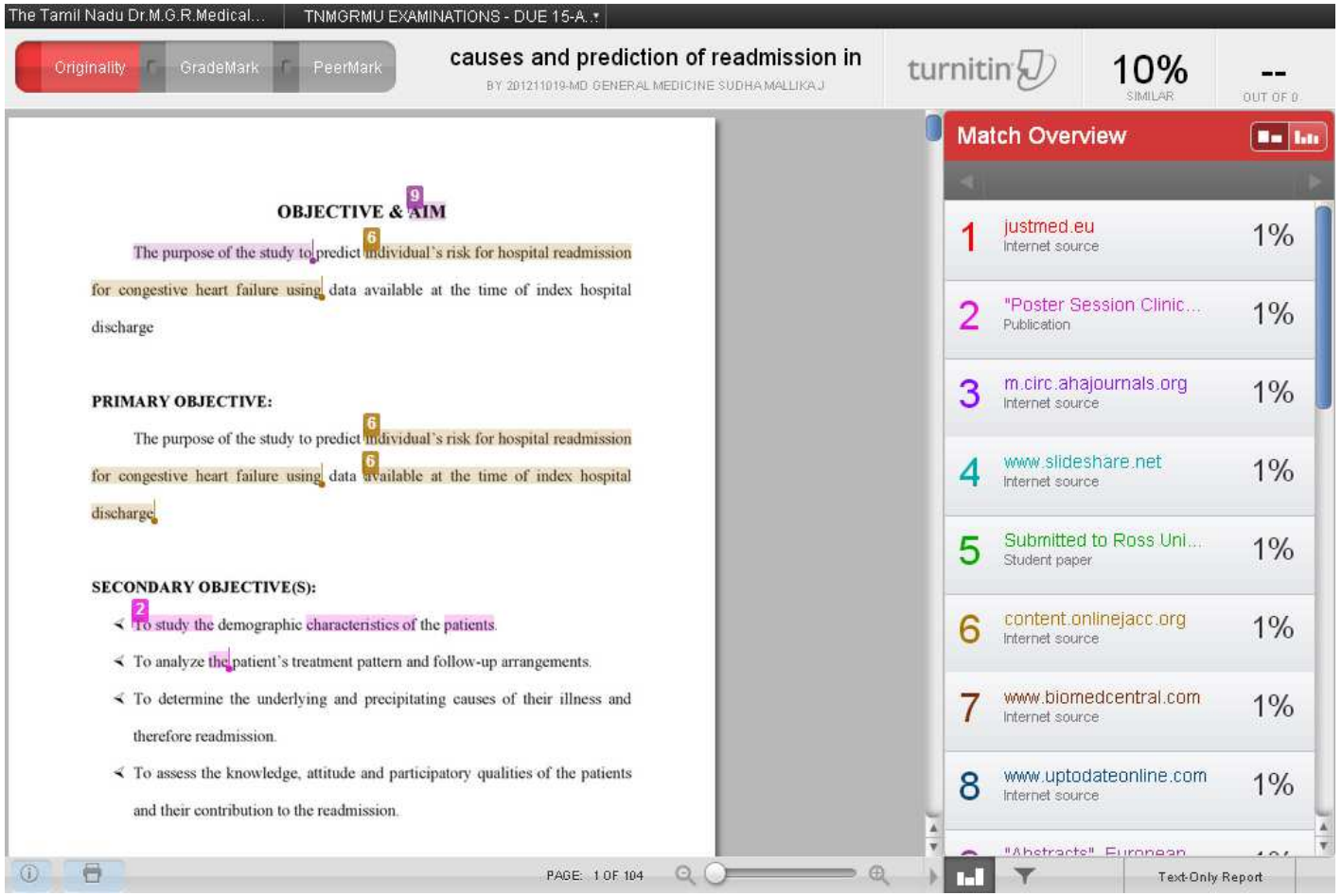
We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003



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OBJECTIVE & AIM

The purpose of the study to predict individual's risk for hospital readmission for congestive heart failure using data available at the time of index hospital discharge.

PRIMARY OBJECTIVE:

The purpose of the study to predict individual's risk for hospital readmission for congestive heart failure using data available at the time of index hospital discharge.

SECONDARY OBJECTIVE(S):

- To study the demographic characteristics of the patients.
- To analyse the patient's treatment pattern and follow-up arrangements.
- To determine the underlying and precipitating causes of their illness and therefore readmission.
- To assess the knowledge, attitude and perioperative practices of the patients and their contribution to the readmission.

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INFORMATION SHEET

We are conducting a study on **“causes and prediction of readmissions of heart failure patients”** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is to asses the**“causes and prediction of readmissions of heart failure patients”** and We are selecting certain cases and if you are found eligible physical examination done. 5ml blood will be collected and 2ml of urine will be collected. You will also echocardiogram, electrocardiogram, chest xray. These tests and special studies do not affect your final report or management.

The privacy of the patients in the research will be maintained sthroughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator Signature of Participant

PATIENT CONSENT FORM

Study title : Causes and prediction of readmissions of heart failure patients

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Name :

Age/Sex :

Identification Number :

Patient may check (☑) these boxes

The details of the study have been provided to me in writing and explained to me in my own language ❑

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research ❑

that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or wellbeing or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study. ❑

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological and biochemical tests. ❑

Signature/thumb impression

Patient's Name and Address:

Signature of Investigator

Study Investigator's Name:

Dr.J SUDHA MALLIKA

Name	Ip No	Sex	Age	Underlying Cause	Comorbid Illness	Precipitating Factor	Duration Of Stay	Cvs	Rs Examination	Ecg	Echo	Chest X Ray	Hemoglobin	Urea	Creatinine	Sodium	Pottasium	Total Billirubin
Kumudha	88924	2	52	1,2	3	1	2	1	1	QS complex v1 to v4,	7	1	8.9	39	1	128	4.3	1
Nagammal	88884	2	40	4,5			2		1	right axis deviation with right and left atrial enlargement	1,3	6	9.9	42	1.3	135	3.2	1.2
Parvathy	87695	2	61	2		8	1	2	1	left atrial enlargement	6	5	10.2	32	1.5	135	2.8	1
Radha	88406	2	58	1,2	3		3		1	ST segment elevation v1 to v4	4,8	1	8.8	16	0.9	139	3.4	0.7
Kanniyammal	88265	2	55	4,5		7	1		1	atrial fibrillation with right axis deviation	1,3	6	9.7	27	1	140	4	1
Violet	89851	2	40	4	4	3	3		1	atrial fibrillation with right axis deviation	1,3	6	11	30	0.8	142	4.2	4
Buvaneswari	89115	2	36	4,5			2	1	1	st segment elevation v1 to v4	1,3	6	11.1	43	1.4	132	4.4	1
Valli	88150	2	40	4	4		1		1	right axis deviation with right and left atrial enlargement	1,3	6	10.5	10.5	0.9	141	4.3	0.9
Anbumani	81437	1	56	2			3		1	poor progression of r wave from v1 to v6	5	5	11.1	30	1	138	4.1	0.8
Marudhamuthu	81246	1	80	1,2	1		2	2	1	QS complex v1 to v4,	6	2	8	56	2.1	140	5.5	0.9
Algappan	78771	1	64	2		5	1	1	1	poor progression of r wave from v1 to v6	7	1	11.2	28	1.2	140	3.4	0.8
Perumal	78740	1	46	2	2		3		1	left axis deviation, left atrial enlargement	5	1	12	30	1	135	4	1
Selvaraj	78497	1	65	1,2	3	6	1	2	1	left ventricular hypertrophy with strain pattern	6	2	13	54	1.8	136	3.8	2.6
Lakshmanan	76148	1	32	2	1	7	2		1	t wave invertion with q waves in v1 to v4	8	1	6.7	212	5	146	5.5	1.3
Selvam	86551	1	45	1,2	3	1	2		1	poor progression of r wave from v1 to v6	7	2	16.9	23	1	140	4	1
Gopi	55054	1	60	2			2	1	1	QS complex v1 to v4,	7,9	3	14	56	2	138	3.8	0.7
Meganadhan	49684	1	62	2		8	3		1,2	left ventricular hypertrophy with strain pattern	7,9	2	11.8	64	1.8	136	3.6	0.8
Varadharamanujam	47194	1	75	1	1,3		3	1	1,2	left axis deviation, left atrial enlargement	7,9	3	10.9	44	1	133	3.5	0.9
Elumalai	86870	1	85	2		8	3		1	left ventricular hypertrophy with strain pattern	5,	1	11.5	27	1	141	4.3	1
Velan	87724	1	44	1	1,3	1	2	2	1	left axis deviation, left atrial enlargement	4	8	10	108	4.1	121	5.2	1.2
Duraisamy	87801	1	52	1,2	1	7	1		1	QS complex v1 to v4,	7	1	9.8	105	2.9	140	3.3	1.1
Saravanan	86611	1	40	4,5	1		3		1	left ventricular hypertrophy with strain pattern	4	6	9	211	2.2	124	3.5	1
Kuppan	86559	1	59	1	3		3	1	1	QS complex v1 to v4,	7,9	8	8	47	2.6	138	4.7	1.3
Christophar	88592	1	59	2	1		2	1	1	poor progression of r wave from v1 to v6, and left atrial enlargement	7,9	1	14.3	25	1.1	140	4	2
Asokan	85385	1	60	3	1	1	2		1,2	poor progression of r wave from v1 to v6	7	1	11	30	1	138	3.8	0.9
Chandrasekar	86254	1	47	3	3		1		1	poor progression of r wave from v1 to v6	5,1o	3	10.2	32	1.3	140	4	1

Name	Ip No	Sex	Age	Underlying Cause	Comorbid Illness	Precipitating Factor	Duration Of Stay	Cvs	Rs Examination	Ecg	Echo	Chest X Ray	Hemoglobin	Urea	Creatinine	Sodium	Pottasium	Total Billirubin
Murugan	83637	1	53	1		1	1			left ventricular hypertrophy with strain pattern	4,5	1	11	30	1	132	2.3	1
Vijayan	82253	1	65	1,2		1	1			poor progression of r wave from v1 to v6	5,8	1	13	28	0.8	140	3.2	0.9
Mani	87033	1	56	2		7	2	1	1	bifascicular block	7,9	1	10.2	28	1.07	140	6.3	0.8
Moorthy	86680	1	45	4		1	1	1	1	atrial fibrillation	7,9	6	10.4	65	1.4	134	6.5	1
Ammu	86633	2	35	2	4		2		1	right axis deviation with right and left atrial enlargement	8	2	11	30	1	136	4	1.6
Parvathi	87710	2	61	2			1			left axis deviation, left atrial enlargement, intraventricular conduction block	8	1	10.5	23	1	136	5.5	1.2
Kanniyamma	87811	2	45	4		5,7	1			double qrs complexes, himalayan p wave	10	7	9.7	32	1.5	139	4.9	1
Meena	87567	2	35	4,5		3	3		1	right axis deviation, atrial fibrillation	1,3	6	13	42	0.8	140	5	1
Arumugam	87717	1	48	2		7	2	1	1	left axis deviation with left anterior fascicular block, poor progression of r wave	5	1	11.5	65	1.2	137	5	0.9
Dhanasekar	85556	1	40	1	3		1		1	left axis deviation , poor progression of r wave	7,10	1	10	26	1.4	146	6	0.8
Rasu	87753	1	63	1,2	3		2			left axis deviation , poor progression of r wave	6	8	12.4	17	1	140	3.2	2
Yashodammal	87695	2	80	1,2			2			left axis deviation with left anterior fascicular block, poor progression of r wave	8	1	11.6	15	1.2	147	4.3	1
Vanaja	87231	2	63	2	3		1		1	poor progression of r wave from v1 to v6	7,9	1	7.4	49	1.3	126	4.3	0.9
Murugan	87198	1	46	2	3		2		1	poor progression of r wave from v1 to v6	8	1	11	30	0.9	130	4	0.8
Sarasu	87398	2	60	3		1	2	1	1	incomplete RBBB, right axis deviation	2,3	7	12	15	0.8	138	4.1	1
Amaravathy	86966	2	75	2			1			right axis deviation with right bundle branch block	3	2	12.5	16	0.6	121	4	1
Mariammal	86547	2	55	2	3		1		1	poor progression of r wave from v1 to v5	8	1	11.1	47	1.3	130	4.2	0.8
Suseela	86998	2	47	2	3	1,7	1			left ventricular hypertrophy with strain pattern	5,	1	8.9	26	0.9	136	3.5	0.9
Jeevarathinam	85673	1	55	2,4	3		2		1	left ventricular hypertrophy with strain pattern	1,4	6	9.2	63	1.2	140	4.1	0.8
Ammaponnu	86790	2	63	4,5			1	2	1	left ventricular hypertrophy with strain pattern, right ventricular hypertrophy	1,4	6	7.4	56	1.1	130	5	2
Gangadharan	85560	2	45	2	3		2	1		poor progression of r wave from v1 to v4	7,9	1	9	30	0.6	134	4	0.9
Umasankar	79485	1	45	2		6	2	1	1	left anterior fascicular block, poor progression of r wave	7,9	8	11.2	27	1.1	130	4.1	1
Saravanan	82233	1	37	1,2	1,3		2		1	left axis deviation with poor progression of	6,9	1	7.8	89	4.6	143	4	0.7
Balakrishnan	87150	1	74	3		1,7	1			left axis deviation with right bundle branch block, poor progression of r wave	7,8	2	12.6	27	1	145	4.1	0.8
Sathik	87095	1	68	3	2	1,7	1	1	1	poor progression of r wave	7,9	3	12.4	27	0.9	143	3.3	0.9
Murugan	87159	1	46	2		4	2		1	left axis deviation, left atrial enlargement, intraventricular conduction block	7,8	2	14	31	1	140	4	1

Name	Ip No	Sex	Age	Underlying Cause	Comorbid Illness	Precipitating Factor	Duration Of Stay	Cvs	Rs Examination	Ecg	Echo	Chest X Ray	Hemoglobin	Urea	Creatinine	Sodium	Pottasium	Total Billirubin
Durairaj	86781	1	53	2	3		1			poor progression of r wave	4,8	1	10.1	24	1.1	143	4.1	2.4
Unnamalai	81251	2	67	2	3		1		1	left axis deviation with right bundle branch block, poor progression of r wave	4,8	1	12.6	30	1.1	138	3.6	1
Venkatesh	86677	1	52	1,2		1,7	2		1	left ventricular hypertrophy with strain pattern, right ventricular hypertrophy	4	1	10.4	30	0.9	132	6	1
Thangaraj	87250	1	40	2	5		2		1	right bundle branch block	5,9	8	10.7	58	2.6	136	4.1	0.8
Subramani	87252	1	36	2			3	1	1	left axis progression , q wave in lead II, III, aVF	7,9	3	9.2	43	1	123	4	1
Yogammal	85675	2	50	2,4	3		2		1	poor progression of r wave	7,1	6	12.4	63	1.2	140	5.6	0.9
Dhanasekaran	85556	1	40	1,2	3		2	1	1	left atrial enlargement,left anterior fascicular block, poor progression of r wave	7,9	3	11	17	1	140	3.2	0.8
Balaraman	85435	1	58	4			2			left atrial enlargement, left ventricular strain pattern	7,1	2	10.4	109	2	140	5.5	0.9
Manikammal	85496	2	58	3	3	8	1			left axis deviation, poor progression of r wave	6	1	13.2	18	0.9	138	3.6	1
Anjalai	85661	2	38	1,2		1,8	2		1	left axis deviation, poor progression of r wave	6	2	12.9	16	0.8	141	4.2	1.8
Amudha	85298	2	50	1,2	3	1,2	1			ventricular premature beats, left ventricular hypertrophy and strain pattern	7	1	7.8	18	0.8	138	5.1	1
Sarala	85286	2	44	4,5	3	7	1		1	left atrial enlargement	5	6	11.8	28	1.1	134	3.2	0.8
Thulasi	82010	2	62	4,5			3	1		atrial fibrillation, right axis deviation	1,6	6	9	15	0.7	139	4.4	0.6
Mariyammal	78990	2	55	3			2		1	poor progression of r wave	3	1	10	30	1	130	3.2	1
Ponnammal	86542	2	65	1,3		1	2	2		low voltage qrs complex,right ventricular hypertrophy	3,7	6	12.4	30	0.8	133	4	0.9
Chithiraithangam	90491	2	82	2	3		2	1	1	left axis progression , q wave in lead II, III, aVF	7,4	3	9.9	25	1.5	134	3.7	0.8
Amutha	90671	2	28	4,5		3	2		1	right axis deviation, atrial fibrillation	1,7,9	6	7.3	28	0.8	130	4.2	0.9
Santhurselvi	90214	2	37	2	2		3			t wave invertion with q waves in v1 to v4	5,8	9	10	28	0.9	110	4.6	0.8
Johnson	88770	1	50	1,2	1	1	3		1	left ventricular hypertrophy with strain pattern, right ventricular hypertrophy	4,8	2	11	271	7.6	132	6.1	0.8
Rajendraachari	87645	1	57	3	4	1	3		1	left axis deviation, left atrial enlargement, st depression	7,9	3	7.3	24	1	135	3.9	0.9
Aru Mugam	90288	1	76	1,2	3		1			left axis deviation	7,9	1	10.6	28	1.3	130	4.6	1
Jayapal	90655	1	54	1,2	1,2	7	3		1	right axis deviation, clockwise rotation	7	2	7.9	85	1.6	140	5	1
Jagadeeswaran	91141	1	55	4,5		3	2		1	right axis deviation, clockwise rotation	1,3	6	8	58	1.4	105	5.5	4.1
Kumari	91087	2	27	4,5			3	1	1	atrial fibrillation, ST segment depression v5 to v6	1,3	6	6.9	86	0.8	130	4.4	0.9
Suriya Banu	92769	2	42	4	4	4,7	1		1	normal axis, left atrial enlargement	1,3	6	6.4	30	1	132	3.2	1
Vasantha	92143	2	47	2	3,4	7	1		1	normal axis , ST segment depression in v5 and v6	8	8	7.9	26	1.2	140	3.4	1

Name	Ip No	Sex	Age	Underlying Cause	Comorbid Illness	Precipitating Factor	Duration Of Stay	Cvs	Rs Examination	Ecg	Echo	Chest X Ray	Hemoglobin	Urea	Creatinine	Sodium	Pottasium	Total Billirubin
Simson	91257	1	40	4	1,4,5	1,7	2		1	atrial fibrillation, right axis deviation	1,3	3	12	86	2.6	145	4	0.9
Poovarasan	88545	1	13	4		3,6	2	1	1	left ventricular hypertrophy, left atrial enlargement	1,4	6	13	24	1	134	4.4	0.8
Vasanth Kumar	92499	1	39	3	5	1,7,8	2		1	left atrial enlargement, ,left axis deviation, left ventricular strain pattern	4	3	13.2	61	2	135	3.6	1
Murugesan	93165	1	45	2	1,5	5,7	1	1	1	sinus tachychardia	4	1	11.5	49	1.6	136	3.6	0.9
Balaji	93193	1	70	2	5	1,8	1		1	qs complex in lead II, III, aVF	8	1	14	28	0.8	142	4	0.7
Sevathan	91998	1	64	4	4	1,2	1		1	left ventricular hypertrophy withleft ventricular strain	7,4	2	8.3	32	0.9	138	3.7	0.8
Damodharan	79266	1	18	1		1	3	1	1,2	left atrial enlargement with RBBB,RV strain pattern	4	8	12	53	1.3	133	3.6	0.9
Jegadeesan	91141	1	55	4	5	1,6	2		1	atrial fibrillation with rapid ventricular rate with right axis deviation	4	6	7.9	58	1.4	140	4	3
Ponnusamy	90897	1	60	2	4		2	1	1	right axis deviation, poor progression of R wave	5,8	1	3.3	49	1.7	132	4	0.7
Muthukani	89886	1	95	3	4		2	1	1	normal axis, left atrial enlargement	4	1	3.4	40	1	134	3.4	0.8
Devan	90817	1	65	2,4	4	3	2		1	atrial fibrillation with QS complex v1 to v3	1,8	6	7	31.4	1.17	140	4.4	0.9
Palani	88880	1	55	3	4	7	2	2	1	sinus tachychardia	4	1	4.4	22	1.2	136	3.8	1
Valli	88636	2	60	2	4	1	2	1		ST segment elevation in lead II, III, Avf	5,8	2	8.2	34	2.1	140	2.4	1
Arumugam	88393	1	46	1	3	7	2		1	sinus tachychardia	4	8	10	28	1.8	140	3.2	1
Chinnappan	88150	1	50	2	5	4	3	1	2	ST segment elevation in lead II, III, Avf	5	3	9.6	34	2.4	130	4.1	1
Rani	87906	2	57	2	3	1	2	1	1	q waves in I, Avl,V5,V6	5	8	13	48	0.9	144	3.8	1.2
Amudha	87663	2	42	4	4	7	3		1	sinus tachychardia	4	1	6.7	31	1.8	134	4	1
Mamimaran	87420	1	51	1	5	8	2			biventricular hypertrophy	4	1	7.4	25	2	138	3.2	1.2
Govindhammal	87176	2	32	2	3	1	3	1	1	qs complex in lead I,AVL,V1,V2	6,9	3	8	34	0.8	140	4	0.8
Lakshmi	86933	2	43	1,4	4	7	3	2	1,2	left ventricular hypertrophy with strain	5,4	6	5.6	42	1.8	140	2.4	0.8

Key to Master Chart

SEX:

Male – 1

Female – 2

UDERLYING CAUSES

Hypertensive heart disease -1

Ischemic heart disease-2

Other cardiomyopathies-3

Valvular heart disease-4

Atrial fibrillation or flutter-5

COMORBID ILLNESS

Acute or chronic renal disease-1

Chronic lung disease:-2

Diabetes mellitus:-3

Anemia:-4

Drug or alcohol abuse-5

PRECIPITATING FACTOR

1. lack of compliance
2. uncontrolled hypertention
3. cardiac arrhythmias
4. inadequate therapy
5. pulmonary infection
6. emotional stress
7. inappropriate medication or fluid overload
8. myocardial infarction
9. endocrine disorders

DURATION OF STAY

1. 1-4 days
2. 5-9 days
3. >10 days

CARDIOVASCULAR SYSTEM AUSCULTATION

1. S3 heard
2. Systolic murmur heard

RESPIRATORY SYSTEM AUSCULTATION

1. Basal crepitations
2. Wheeze

ECHOCARDIOGRAM

1. Valvular disease
2. Interatrial and interventricular shunts
3. Right ventricular dilation and D shaped interventricular septum
4. Left ventricular diastolic dysfunction
5. Left ventricular systolic dysfunction EF; 41- 45%
6. Left ventricular systolic dysfunction EF; 36-40%
7. Left ventricular systolic dysfunction EF; 30-35%
8. Regional wall motion abnormality
9. Global hypokinesia
10. Pericardial disease

N; CHEST X RAY

1. Pulmonary hypertension grade I according to larry Elliot’s classification upper lobe veins prominent

2. Grade II either kerley A,B,C lines or hilar haziness
3. GradeIII- bilateral patchy opacities
4. Grade IV- bilateral milliary mottling
5. Pleural effusion
6. postcapillary pulmonary arterial hypertension showing main pulmonary artery dilation
7. precapillary pulmonary arterial hypertension
8. normal lung field
9. suggestive of COPD

BILLIRUBIN

1. Elevated
2. Not Elevated